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The Horse's Heart and the "Hamama" Case

IN this issue of THE VETERINARY RECORD is published an abbreviated account of the salient features of the much discussed "Hamama Case," recently heard in the High Courts. The text of the final Judgment, except for purely legal arguments, will follow next week. It has been felt that there are issues in this case of importance to members of the profession, and it is for that reason we are dealing with it at length. A considerable responsibility devolves upon a veterinary surgeon who is asked to provide a certificate of soundness for a domesticated animal of whose past history he may well know little or nothing. The public, and perhaps especially the horse-owning public, has developed a great measure of confidence in a veterinary certificate of soundness, and this is a reflection of the degree of honesty, conscientiousness, and ability with which members of our profession have carried out these examinations over past years. To build up a reputation for reliability in this way is itself a very great achievement, and one which we must guard and strive to maintain.

A complete cardiological examination is neither expected nor possible at an examination for soundness, as carried out under normal circumstances by a veterinary surgeon in general practice. On the other hand, no veterinary surgeon to-day would examine a horse without making a careful auscultation of the heart, at rest and after both reasonable and severe exercise such as will put the heart under both mild and more considerable stress. He will also assess the character of the pulse beat under these conditions. By such methods, conscientiously carried out, the great majority of the symptoms of cardiac errors and abnormalities to which a horse is liable will be revealed, although their exact interpretation may be very much more difficult.

One of the most important characteristics of normal cardiac function in any mammal is an inherent regularity of rhythm. The mechanical precision of the component intervals of the cardiac cycle, when measured by exact timing mechanisms, is, to say the least, extraordinary. The human ear is quite unable to detect variations which may measure only one or two twenty-fifths of a second; but the more serious arrhythmias are of greater magnitude than this, and where partial heart block ("missed beats") occurs, whether regular or irregular, it becomes very difficult to make an interpretation of their exact significance. Many veterinary surgeons, when examining a mature horse (6 years old or more), which has a big powerful heart are inclined to disregard as of no consequence a "missed beat" of this nature, provided it disappears after exercise. They may decide not to make any mention of this in the certificate they furnish subsequently. In some cases they are probably correct, since there seems good evidence that a hypertrophied but normal heart may be functionally capable of providing adequate blood circulation during periods of rest by contracting perhaps only 5 times during a period when otherwise 6 contractions would normally occur. Such a "missed beat" is a functional adaptive economy on the part of the heart. When demand for even a little extra blood occurs (exercise) the regular rhythm is immediately established and no missed beat is shown.

On the other hand, there is no doubt that the *normal* physiological method of adjusting the cardiac output of blood to meet the body's immediate demands is by a change in the frequency of the heart beat without any alteration in heart rhythm; the heart beat slows down at rest and during sleep, and speeds up under the influence of exercise, fear, excitement, etc.

Partial heart block may have different causes, the most usual being some minor (or major) interference in the conducting mechanism (Bundle of His), and the detection of a minor abnormality in conduction is fraught with major difficulty.

It is logical to conclude that any and every "missed beat" or other alteration in rhythm is an abnormality which must be noted in the certificate, even though, in the veterinary examiner's opinion, it is most unlikely to incommode the horse in any way or to lower its efficiency for the purpose for

which it is to be used. He is entitled to his opinion, honestly reached, and even though he may eventually be proved wrong the fact that he mentioned the irregularity absolves him from any question of carelessness or omission in his examination.

This is an instance of only one of the complications which may arise. With reference to abnormal sounds, murmurs, right/left asynchrony, and many other abnormalities, the veterinary surgeon is faced with almost equally difficult decisions, but again to safeguard himself he should invariably make mention of them in his certificate, qualifying them if necessary, and if in any doubt should constantly bear in mind that while many horses with cardiac abnormalities are able to hunt or even race successfully, the rider's life may be at hazard if the horse has a doubtful heart. Many a horse has died from heart syncope, while hunting, chasing, or racing, and many more have been pulled up or eased up just in time to avoid a serious fall.

In the case of "Hamama," there was no veterinary certificate furnished before the horse was purchased, although the purchaser and his agent were under the impression that such a certificate was in existence and would be forthcoming. The majority of those who purchase expensive race-horses make it a rule to do so subject to a veterinary examination, and in future it seems likely that, with the example of the "Hamama" case in mind, more emphasis may well be put upon the actual certificate than upon verbal statements regarding the soundness of a horse. It is also possible that purchasers may expect the veterinary surgeon to pay perhaps a little more attention to heart action and to any unusual features encountered.

There are thus two lessons to be learnt from the "Hamama" case: first, for the purchaser, to ensure that before he completes a purchase he has the actual certificate of soundness; second, for the veterinary surgeon, that he continues to exercise all care when examining a horse for soundness and does not dismiss abnormalities in heart action too lightly.

It is not suggested that any new principles have emerged from the hearing of the case or from the Judgment, but readers will note Mr. Justice Paull's comments:—

> "You do not warrant that a horse has a heart which will remain sound during the horse's life, or that the heart is so strongly built that it is unlikely to break down at some future date. You warrant that at the moment of sale the heart is not then diseased and that the heart is fit for work of the kind to which the vendor has reason to believe the horse may reasonably be put in the immediate future."

And later:—

> "One veterinary surgeon may examine and pronounce the heart normal; another may examine and pronounce the heart a strained heart, yet each may be right on what his examination has shown. It is clear that the subject bristles with difficulties."

These comments are important and may well be referred to in any future legal actions concerned with soundness of the horse's heart.

Nematodirus helvetianus, an Intestinal Worm of Cattle

BY

J. H. ROSE

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SUMMARY.—Nematodirus helvetianus *is briefly described. Illustrations are provided which should facilitate the identification of the* 4 *species of Nematodirus which have been recorded from British cattle.*

NEMATODIRUS helvetianus was first described by May (1920) in the U.S.A. It would now seem to be the commonest species of Nematodirus found in American cattle. It is, in fact, the only species of this genus included by Douvres (1957) in his list of the nematodes most commonly found in the gastro-intestinal tract of cattle in various representative regions of the U.S.A.

In this country, *N. helvetianus* was recorded for the first time only a little more than 2 years ago by Morgan and Soulsby (1956). It was recovered, with other nematodes, from cattle suffering from parasitic gastro-enteritis on farms in the south-west of England. This nematode was recently observed at Weybridge while examining material which had been collected in the course of the routine *post-mortem* examination of cattle sent to the laboratory from farms in the south-east of England.

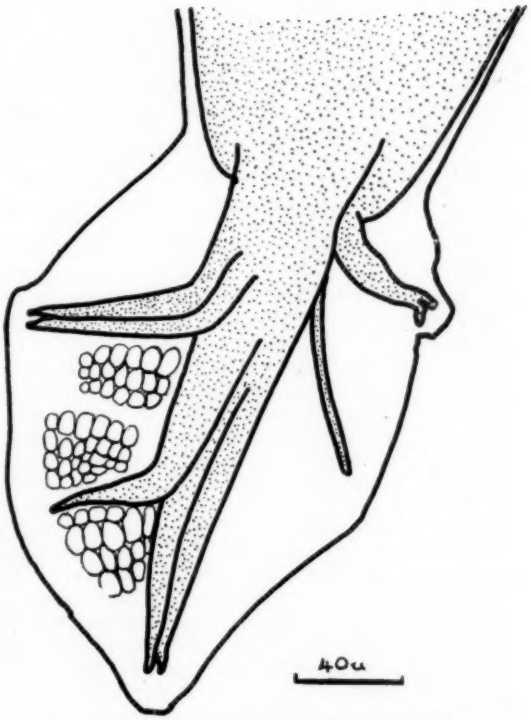
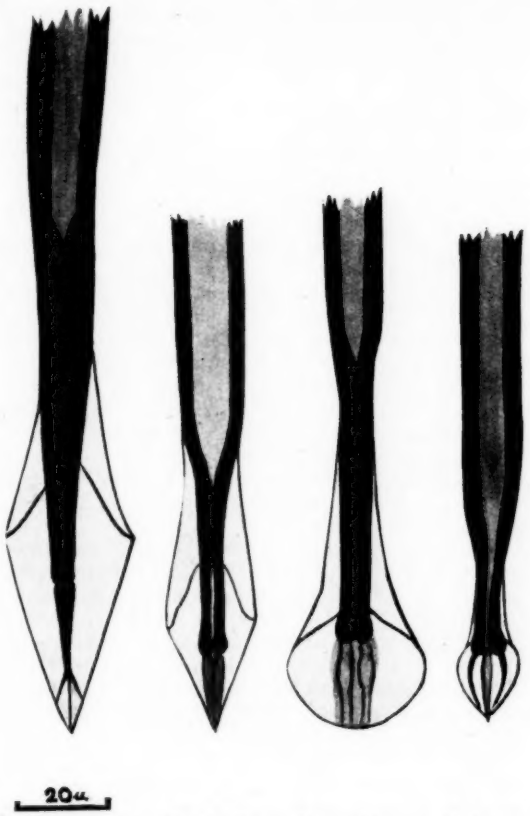


FIG. 1.—*N. helvetianus.* Bursa of male, lateral view.



N. helvetianus. *N. filicollis.* *N. spathiger.* *N. battus.*

FIG. 2.—Posterior end of spicules.

It seems likely that *N. helvetianus* may be more prevalent in our cattle than is realised. As there does not appear to be any readily available description of this nematode, in this country, a brief illustrated description of those features used in the identification of the worm, all of which are easily seen on microscopical examination of unstained specimens, seems appropriate.

Description of *Nematodirus helvetianus*

The male worm is small and slender, reaching 17 mm. in length. The bursa is relatively small; its general shape, together with the arrangement of the bursal rays, as seen from the lateral aspect, is illustrated in Fig. 1. The spicules are long and slender, and are united by a membrane which terminates posteriorly in a somewhat spear-shaped process as illustrated in Fig. 2.

The female worm may reach 25 mm. in length. The anterior end is slender but the posterior region is thickened. The tail is truncated and bears a short spine (Fig. 3). The eggs, which are readily seen in the mature female, are large and oval, measuring 150 to 230 μ long by 85 to 110 μ wide (Fig. 4).

The cuticle at the anterior end of both male and female worms is inflated and striated.

Differentiation of Species

Four species of Nematodirus have been recorded from the small intestine of the bovine animal in this country, namely: *N. spathiger, N. filicollis, N. helvetianus,* and *N. battus* which was recently recorded from cattle for the first time by Parfitt and Michel (1958).

The male worms of these 4 species differ from one another in the form and size of both the bursa and the spicules. They can be differentiated readily, however, solely by reference to the posterior end of the spicules as will be evident from Fig. 2. It can be seen that the size and shape of the posterior termination of the spicules is characteristic for each species. In the case of *N. helvetianus* and *N. filicollis,* the terminations are similar in outline; they do, however, differ considerably in both size and structure, and in practice they can be differentiated readily.

Examination of the spicules is not as difficult as might be supposed. They are frequently partially

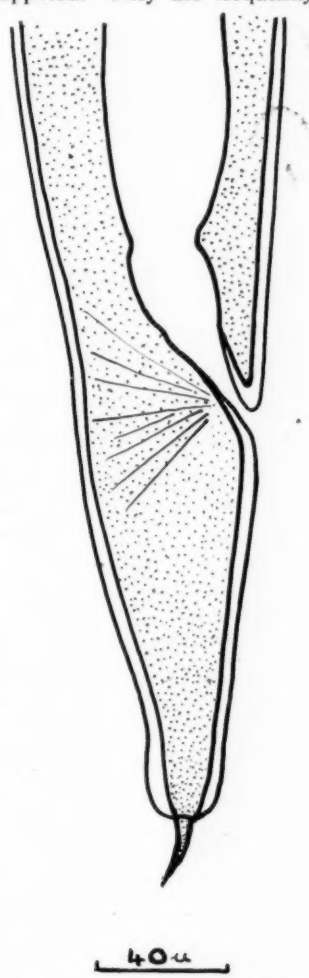


FIG. 3.—*N. helvetianus.* Tail end of female.

extruded, and when the worm is mounted on a microscope slide, under a cover slip, the spicules can be orientated into the dorsal or ventral plane by gently moving the cover slip. The terminal region can then be compared with the illustrations.

The female worms, except for *N. battus*, are more difficult to differentiate. The female *N. battus*, which has a conical tail tapering to a fine point, has been well described and illustrated by Crofton and Thomas (1954). The female worms of the other 3 species all have a truncated tail bearing a short spine. *N. battus* can, therefore, be differentiated by the tail alone. The eggs also have a distinctive shape (Fig. 4) and provide another aid to identification. The size and shape of the egg also provides a means of differentiating *N. filicollis* from *N. spathiger* and *N. helvetianus*, but as can be seen from Fig. 4 the eggs of *N. spathiger* and *N. helvetianus* are very similar and cannot be used as a means of differentiation. The female worms of these two species are so similar that unstained specimens cannot be differentiated readily.

Discussion

The nematodes of cattle have been the subject of a considerable amount of investigation in the U.S.A., and it can be taken for granted that the recognition of *N. helvetianus* as one of the nematodes commonly found in the gastro-intestinal tract is based on adequate evidence. In this country our knowledge of cattle nematodes is much more limited.

Very little is known about the incidence of the different species of Nematodirus in British cattle. *N. spathiger* is thought by some to be the species most often found in our cattle. This belief would seem to have its origin in Monnig's (1956) text-book of Veterinary Helminthology and Entomology, in which he states that *N. spathiger* is the commonest species (of Nematodirus) in sheep, cattle and other ruminants. It should be noted, however, that there is no specific reference to British stock. On the other hand Lapage (1956) states that *N. filicollis* is the species most often found in British sheep and cattle, whereas he lists *N. spathiger* as occurring only in sheep.

The only other available information regarding the incidence of Nematodirus spp. appears to be that obtained from the results of routine *post-mortem* examinations of cattle suffering from parasitic gastritis. In the course of such work the species of Nematodirus present may be identified by reference to female worms only as the features used for specific identification are considered to be more readily observable in the female than in the male worm. It has been shown, however, that the female *N. helvetianus* cannot be differentiated readily from the female *N. spathiger*. It seems possible, therefore, that *N. helvetianus* has frequently been mistakenly identified as *N. spathiger*. When male worms have been identified it is possible that *N. helvetianus* has been mistaken for *N. filicollis*, as the posterior termination of the spicules is so similar in these two species. When it is borne in mind that up till now a description of *N. helvetianus* has not been widely available in this country, it will be realised that such mistakes could readily be made. Information from routine *post-mortem* examinations is therefore unlikely to be at all reliable.

Having regard to the incidence of *N. helvetianus* in American cattle, the observations of Morgan and Soulsby (1956) and observations made at Weybridge, it is suggested that this species of Nematodirus is not as rare in this country as present records might suggest. It is hoped that this paper will aid in the proper identification of the 4 species of Nematodirus which have been recorded from cattle, and that eventually accurate information on the incidence of the different species will become available.

References

CROFTON, H. D., & THOMAS, R. J. (1954). *J. Helm.* **28**. 119.

DOUVRES, F. W. (1957). *Amer. J. vet. Res.* **18**. 81.

LAPAGE, G. (1956). "Veterinary Parasitology." Oliver & Boyd, London. P. 69.

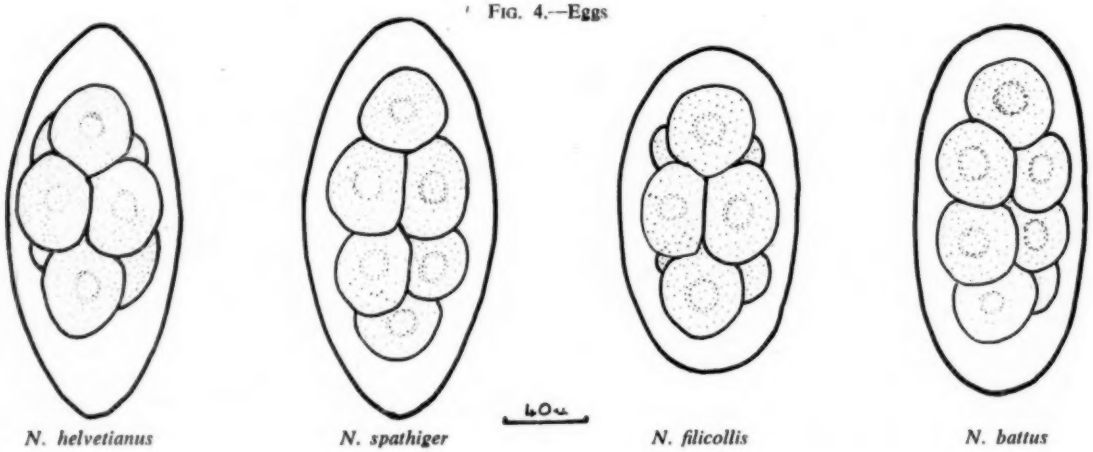
MAY, H. G. (1920). *Proc. U.S. Nat. Mus.* **58**. 577.

MONNIG, H. O. (1956). "Veterinary Helminthology and Entomology." 4th edition. Baillière, Tindall & Cox, London. P. 227.

MORGAN, D. O., & SOULSBY, E. J. L. (1956). *Vet. Rec.* **68**. 1,029.

PARFITT, J. W., & MICHEL, J. F. (1958). *Ibid.* **70**. 71.

FIG. 4.—Eggs



N. helvetianus *N. spathiger* *N. filicollis* *N. battus*

Biochemical Aspects of Bovine Ketosis*

BY

J. Y. F. PATERSON

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THIS paper will describe briefly a programme of work which was thought might shed some new light on the problem of bovine ketosis. When this work was begun there were several views on the aetiology of ketosis, but in the large volume of published work on this subject there was no unequivocal support for any one of these hypotheses. It seemed that one line of investigation which might possibly yield new evidence would be a study, by biochemical analyses, of the course of development of bovine ketosis.

This had been done once before by J. C. Shaw (*J. Dairy Sci.* 1943. **26.** 1,079) who studied animals in a herd with a very high incidence of ketosis. He determined blood glucose and ketone bodies, and also liver glycogen by the increase in blood glucose following adrenalin injection. In animals which developed ketosis it was found that blood glucose levels began to decrease and ketone body levels to increase a few days after calving, and at 1 to 2 weeks after calving there was marked hypoglycaemia and ketonaemia.

In these animals liver glycogen was less than normal. The external symptoms of ketosis, inappetence, weakness and decreased milk yield appeared when the animals were hypoglycaemic.

For our investigation no herd with a high incidence of ketosis was available, but in any case ketosis at the herd level can often be traced to a dietary cause, or at least corrected by alteration of the diet. Individual spontaneous cases of ketosis in otherwise normal herds had therefore to be sought, and the most likely animals were those which had had ketosis in the previous lactation. A group of such cows was selected, and also a group of normal cows of the same age, on the same farms, and calving at the same time as the previously ketotic cows. However in this first part of the study no cases of ketosis occurred on the farms used, and so the study was one of normal animals only. The results have been published (*J. comp. Path.* 1957. **67.** 165, 331) and briefly were as follows :—

(a) There is little 17-hydroxycorticosteroid in the blood of dairy cows, the level being more or less insignificant until 1 to 2 weeks *pre partum* when higher blood levels of steroid were found in most cows. Similar results have been obtained recently by Brush (*J. Endocrinol.* 1958. **17.** 381). The changes in leucocytes were as expected.

About one-third of the cows showed transient increases in 17-hydroxycorticosteroids in the period 2 to 4 weeks *post partum*.

(b) Blood glucose increased just before calving, as has been found by earlier workers. It seems possible that this increase in blood glucose may be related to the increase in plasma 17-hydroxycorticosteroids. About two-thirds of the cows showed a decrease in blood glucose in early lactation, ca. 2 to 4 weeks *post partum*, and in a number of these, surprisingly low levels of glucose were seen. There was no ketonaemia associated with this hypoglycaemia, but only acetone and acetoacetic acid were estimated.

In the second part of the investigation 7 cows with previous histories of ketosis were used. Of these 4 remained clinically normal and all 4 showed the blood changes seen in the majority of animals in the first stage of the work. The blood level of glucose increased about 1 week before calving and this increase was coincident with an increase in plasma 17-hydroxycorticosteroids. About 2 to 4 weeks after calving each cow showed a transient hypoglycaemia, and this time significant ketonaemia was also found. The difference between this observation and our earlier observation is accounted for by β hydroxybutyric acid which was estimated in this second part of the investigation. There was also at this time a transient increase in plasma 17-hydroxycosteroids.

I feel that this transient hypoglycaemia in early lactation is a significant observation. There is a seasonal variation in the blood level of glucose in normal cows, the lowest levels being found in the

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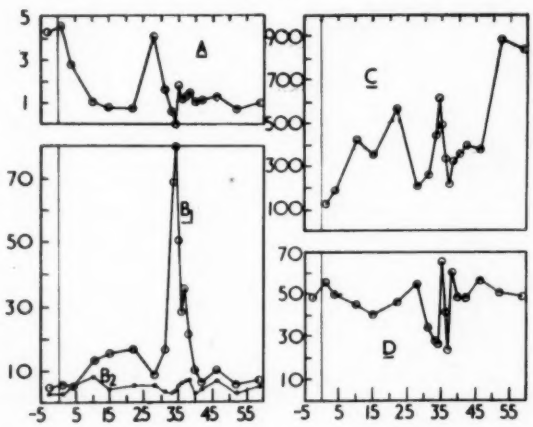

FIG. 1.—Blood components in a cow during the development of ketosis, and treatment with corticotrophin (160 I.U. administered on 34th and 37th days). A, 17-hydroxycorticosteroids (μg. per 100 ml. plasma); B_1, total ketone bodies (mg. per 100 ml. blood); B_2, volatile fatty acids (mg. per 100 ml. plasma); C, eosinophils (cells per c.mm.); D, glucose (mg. per 100 ml. blood).

* Paper presented to the Conference of the A.V.T. & R.W. at Gleneagles, April 2nd-3rd, 1959.

Tuberculosis of Animals and its Transmission to Man

BY

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SUMMARY.—It is shown that bovine-type infection does not persist as an epidemic in any species but cattle, unless it is constantly renewed from a bovine source.

The spread and control of bovine tuberculosis is very briefly reviewed. Early acts from 1885 onwards gave little or no control over the safety of milk and the Tuberculosis Order of 1909 was not effectively introduced until 1925. By 1905 the work of the British Royal Commission had clearly shown the danger of bovine-type infection to man but it was not until the 1930s that there was any strong demand for pasteurisation, and only in 1949 that regulations for the compulsory pasteurisation of milk were introduced.

The Attested Herds Scheme was introduced in 1935 and the Area Eradication Plan for Tuberculosis in 1950. There are now (March, 1957) 7.4 million cattle in Attested Areas or Areas undergoing eradication, out of a total cattle population of some 9 million.

IN another article (Francis, 1959) the writer reviewed the work of the British Royal Commission (1901-11) which clearly established that bovine tubercle bacilli were pathogenic for man. It was many years, however, before effective action was taken to prevent infection of man by bovine tubercle bacilli. Now that effective measures are being taken it is of some interest to describe briefly the ways in which man may become infected and the methods that have been adopted to prevent infection.

Man may occasionally be infected with the human type of tubercle bacilli from monkeys, dogs, elephants, or parrots. He may be infected with the bovine type by direct contact, or by meat products, from a variety of species. It is, however, an interesting and important fact that bovine-type infection does not persist as an epidemic in any other species, including of course man, unless it is constantly renewed from a bovine source. If, therefore, we exclude avian tuberculosis, which is very seldom transmitted to man, it will be seen that we may concentrate attention almost entirely on tuberculosis of cattle (Francis, 1958).

Bovine tuberculosis probably spread from the Mediterranean littoral through Switzerland, Germany, and Holland to Great Britain, and the town dairies of the industrial revolution provided ideal conditions for its spread. The Scandinavian countries were infected by cattle from Britain and Holland and by the middle of the nineteenth century European and British cattle were heavily infected. During that century and the next the disease spread to the countries of the New World largely due to the extensive importation of British cattle.

Prevention of Human Infection with Bovine Bacilli

It has been stated (see Francis, 1947) that there were rules against the consumption of tuberculous flesh in the Mosaic laws. Villemin's experiments on infectivity were reported in 1868, and an idea of enlightened opinion concerning tuberculous milk and flesh in 1875 can be given by quoting from Fleming's "Veterinary Sanitary Science and Police." He says: "Any organ or texture in which tubercle is deposited, as well as tubercular matter of any description, should not be considered fit for

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Biochemical Aspects of Bovine Ketosis—*Concluded.*

period November to February, and it was in this period that the most marked *post partum* hypoglycaemia was seen. Thus at a time when ketosis is commonly prevalent, early lactation in mid-winter, the normal cow may show a marked hypoglycaemia and ketonaemia of short duration. There is no evidence of the cause of these blood changes, but it seems important that they should be verified and studied further, since they may indicate that ketosis is the result of accentuation or prolongation of normal changes.

In this second part of the investigation, 1 cow developed typical clinical ketosis, and results from this animal are shown in Fig. 1. Until the 28th day after calving the cow was quite normal, but on the 31st day showed slight hypoglycaemia and ketonaemia. On the 33rd day the hypoglycaemia and ketonaemia were very marked, and the cow now showed other symptoms of ketosis—refusing concentrates, decreased milk yield, etc. On the 24th day the animal's condition was unchanged and 160 I.U. ACTH was given. This corrected the hypoglycaemia and substantially reduced the ketonaemia and on the 36th day the cow had improved somewhat. But next day her condition had worsened and a further 160 I.U. ACTH was given. Following this there was a rapid and uneventful recovery. In the case of this animal the inappetence and decreased milk yield appear to have followed the development of hypoglycaemia.

However one case does not form a basis for discussion, but these results do show the type of results that it was hoped might be yielded in this investigation. It is unfortunate that during the period of study there was very little ketosis among the herds investigated, but in spite of this failure, I feel that this approach to the problem of ketosis is worth further efforts and might still yield new information.

food. As we have no proof that the bones or muscles are usually the seat of tubercle, unless, perhaps, in very advanced cases of the disease, these may be utilised if otherwise in a healthy condition. . . . Of course, the flesh of cattle in an advanced stage of Phthisis must not be consumed, as, in addition to the risk of its unhealthiness, it is of very inferior quality. Everything must depend upon the extent of the alterations and the gravity of the tubercular lesions. When the tubercles are soft and there are purulent masses, and the alterations are otherwise numerous, danger is to be apprehended from the consumption of the flesh as food. On the contrary, so long as the tumours are hard and dry and no cascous infiltration has taken place, and especially when the majority of the organs in the abdomen and thorax are not invaded, then the risk is less." This statement is in exact accord with modern views on meat inspection (see Francis, 1958).

Concerning milk, Fleming quotes experiments showing that the feeding of tuberculosis milk may produce the disease and says: "There is every reason, then, to prohibit the use of milk from cows affected with Tuberculosis, and especially for infants, who mainly rely upon this fluid for their sustenance, and whose powers of absorption are very active." He realised that boiling milk destroyed its infectivity.

Regulations governing the safety of milk were reviewed by Francis (1957). From 1885 onwards, various Acts and Orders were made but they gave little control over the quality of milk, being chiefly designed to protect the public from fraud. Thus in 1894, a Select Committee concluded that: "As the law stands at present, all milk, whatever its composition, which is the produce of a cow, and to which nothing has been added, and from which nothing has been abstracted, must be regarded as pure, and its sale is no offence under the Adulteration Acts." The Milk and Dairy Order of 1926 was the first real attempt to improve the sanitary quality of milk, but we are still some way from the ideal of Sir George Brown, who, as Chief Veterinary Officer giving evidence before the Inter-departmental Committee of 1901, described "genuine milk" as "the fluid drawn from a perfectly healthy udder of a perfectly healthy cow living under the most favourable sanitary conditions."

Pasteurisation

Although Pasteur was the first to study scientifically the preservation of liquid foods by moderate heating, the value of heat for this purpose was, of course, known to the ancients. In 1765 Spallanzani observed that boiling preserved meat extract, and in 1782 Scheele preserved vinegar in this way. Pasteur worked on wine from 1862-4 and on beer in 1870. In 1873 Jacoby (New York) recommended cooking to stop the souring of milk, and in 1886 Soxhlet advised the boiling of milk for infant feeding. Theobald Smith in 1899 determined the thermal death-point of the tubercle bacillus in milk as 140° F. for 15 minutes (Davis, 1955).

We learn from the Oxford Dictionary that the word pasteurisation was first used in *The Pharmaceutical Journal* in 1881, and in 1895 the *Westminster Gazette* stated that the "Pasteurisation of beer had become a phrase." In 1897 the *Melbourne Argus* described the pasteuriser as "a circular tank of tin into which the milk is received."

It is of some interest that compulsory pasteurisation was first introduced in 1898, not to protect human health, but to prevent pigs and calves becoming infected with separated milk returned from Danish dairies: a simple chemical test was used to ascertain whether milk had been treated (Bang, 1899, pp. 377 and 397). Apparently this measure never gave rise to any serious controversy. Compulsory pasteurisation of milk for human consumption was introduced in New York in 1907 (*Lancet*, 1907, **i**, 826), but as we have seen, publication of the Reports of the Royal Commission produced little demand for the introduction of pasteurisation in Great Britain. Regulations defining methods of pasteurisation were laid down in 1922 and 1923 (*Lancet*, 1928, **i**, 255), but the index of the *Lancet* reflected only slight interest in the subject during the 1920s. In 1931, there was, however, a burst of 15 entries in the index under "pasteurisation," and few subjects have given rise to more public controversy. Sir William Savage (1931) remarked that compulsory pasteurisation was much under discussion. The main questions were whether it would render milk safe, and the extent to which it damaged its nutritional properties. In 1932, the People's League of Health published "A Survey of Tuberculosis of Bovine Origin in Great Britain." It advocated compulsory pasteurisation. It should be realised that pasteurisation was already widely used by the large dairying companies because without it they are unable to deliver milk to the town consumer without it going sour. They therefore favoured pasteurisation which was a heavy economic burden to small firms, and particularly to the producer retailer. These facts added much heat to the controversy.

The British Milk Industry Bill of 1938 made provision for compulsory pasteurisation but it met with so much opposition that it was withdrawn, and it was not until 1949 that regulations for the compulsory pasteurisation of milk were introduced.

Milk represents a greater danger to man than does tuberculous meat, and Dr. Hope (1901) read a valuable paper to the British Congress on Tuberculosis in which he dealt with the relative value of sterilisation or pasteurisation *versus* the establishment of tubercle-free herds, to protect man from bovine infection. He said: "It is obvious that numerous points are involved in the subject, some of which are difficult to dissociate from questions other than those which concern tuberculosis only. For example, measures taken with the sole object of checking an even more destructive form of disease—*viz.* diarrhoea—have proved incidentally a safeguard against tuberculosis." He realised that pasteurisation prevented a number of other infections, but speaking of tuberculosis he said: "Sterilisation, valuable as it is as a final safeguard against tuberculosis, is, after all, only an expedient, and must not be put into so much prominence that the importance of the other safeguard is lost sight of. . . . Beyond any questions the

ultimate advantage lies in obtaining the milk from herds free from tuberculosis."

This statement would seem to stand as a fair summing-up of the position, particularly as we now appreciate more fully the danger of direct air-borne infection between cattle and man.

Control of Bovine Tuberculosis

This is obviously the most radicle and satisfactory means of preventing human infection and interest in the subject became active during the last quarter of the 19th century. The early pioneers such as Nocard, Bang, and M'Fadyean were quick to realise the value of tuberculin, but the eradication of tuberculosis by the "stamping out" process of slaughter, and application of sanitary science as applied to cattle plague and pleuropneumonia, was regarded as impracticable because of the great prevalence of the disease. Consequently, various clinical and microscopic methods were employed to detect tuberculous cattle; these were largely public health measures but it was hoped that they would lead to an over-all reduction of incidence in cattle. They were applied with great thoroughness in Germany by Ostertag, but with very little effect on the over-all incidence.

It was not until the tuberculin test was applied systematically and reacting cattle segregated or destroyed, that any real progress was made. The first campaign was begun in Finland in 1899. In the United States, eradication under the accredited herd and area plans was begun in 1917 under the direction of Mohler and Kiernan. They first obtained the co-operation of veterinary practitioners and believed that the only way to introduce the scheme in an area was "to hold meetings: and put the matter clearly before the people as to every step it will be necessary to take in order that success may be attained." Eradication was virtually completed by 1935 and the success of this great scheme has been described in a colourful manner by Myers (1939) in his book "Man's Greatest Victory over Tuberculosis." It undoubtedly did much to make people believe in the possibility of eradicating tuberculosis from large cattle populations, and indeed from human communities. Good progress in the eradication of bovine tuberculosis is now being made in nearly all the more advanced countries of the world.

It was only in 1909 that the first Tuberculosis Order was introduced in Great Britain and cows with suspected tuberculosis of the udder had to be reported and destroyed. This order was suspended during the 1914-18 war, and not re-introduced until 1925. Grade A tuberculin-tested milk was recognised under the Milk (Special Designations) Orders, 1923, but this did little to promote a progressive increase in the number of tubercle-free herds. Accordingly the Attested Herds scheme was introduced in 1935, and powers to carry out area eradication were granted in the Agriculture Act of 1937. The area eradication plan for tuberculosis was introduced on October 1st, 1950, and there are now (1957) 7.4 million cattle out of a total of some 9 million cattle in areas from which tuberculosis has been

(*Concluded at foot of adjoining column*)

Tuberculosis of Animals—*Concluded.*

eradicated, or in which this desirable objective has been nearly achieved.

References

BANG, B. (1899). "Nordisk Medicinisk Arkiv," 1899. Reprinted in Selected Works. 366-410.

DAVIS, J. F. (1955). "A Dictionary of Dairying." London.

FLEMING, G. (1875). "A Manual of Veterinary Science and Police." London.

FRANCIS, J. (1947). "Bovine Tuberculosis." London.

———. (1957). Tuberculosis of Animals, in "Symposium on Tuberculosis." Edited by R. F. G. Heaf. London.

——— (1958). "Tuberculosis in Animals and Man: A Study in Comparative Pathology." London.

———. (1959). "Tubercle." **40.** 124.

HOPE, E. W. (1901). *Lancet.* **ii.** 197.

MYERS, J. A. (1939). "Man's Greatest Victory over Tuberculosis." Illinois.

SAVAGE, W. G. (1931). *Lancet.* **i.** 543.

A Case of Tetanus in the Dog Treated with Chlorpromazine

BY

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Introduction

ALTHOUGH tetanus is recognised in all species of domestic animals it is least frequent in the carnivore, seldom being seen in the dog and even less often in the cat. In this country there have been few recorded cases in small animals but in tropical and sub-tropical regions the incidence is higher.

Case History

In March, 1958, a Corgi dog aged 10 years was presented for examination. The owner reported that although bright and active on the previous evening the dog was now unable to walk freely. Examination showed the animal to have a slight dragging action of the hind quarters but to be apparently normal in all other respects. There had been no other deviation from normal behaviour except that no faeces had been passed for at least 24 hours.

A tentative diagnosis was made of spinal disc protrusion and a guarded prognosis was given. The owner was advised to keep the patient quiet, and an intramuscular injection of chlorpromazine* (25 mg.) was given to assist this. Tablets of the same drug were supplied for continuation doses, and in addition the owner was instructed to give the dog a purgative on its return home (Aloes B.P. gr. 5).

On the following day the dog was seen to be in a much worse condition. There was almost complete extensor tetany with marked trismus. External stimuli provoked more severe spasm but there were no periods of complete relaxation. The owner reported that the dog had been unable to bark or swallow since its return home and that none of the

* Largactil: May & Baker Ltd.

(*Continued on page* 477)

chlorpromazine tablets or the purgative had been taken.

Strychnine poisoning was suspected but was ruled out by the absence of periods of relaxation and, later, on the long duration of the symptoms. There had been no history of previous virus infection or systemic disease to account for nervous symptoms. In view of the close similarity of the symptoms to those seen in other species, a diagnosis of tetanus was made. A detailed search for a focus of infection revealed only a surface scratch on the muzzle, believed to be the result of an encounter with a farm-yard cat. The owner was advised of the poor prognosis but requested that treatment be attempted.

Treatment

In view of the favourable reports of the use of chlorpromazine in cases of tetanus in the human subject and in horses it was decided that this was to be the only drug used to control the spasms of tetanus. On the first 3 days of treatment tetanus antiserum and antibiotics (penicillin and streptomycin) were given. The first dose of chlorpromazine was 25 mg. given by intramuscular injection but all other doses were oral. The quantity used was determined by the owner as that sufficient to prevent the return of severe spasms. This was about 200 mg. a day given in 4 or 5 doses. On the second day of medication the dog was seen to be more relaxed but external stimuli could cause a spasm to become severe. Otherwise the dog lay quietly but was still semi-rigid. Respiratory movements were slightly accelerated and deeper than normal, probably due to involvement of the diaphragm, and the body temperature was slightly sub-normal. Small quantities of fluid were given by the owner after each dose of chlorpromazine, using a plastic bottle and tube.

Recovery was slow but uneventful. On the fourteenth day the owner reported a return of the desire to drink and voluntary swallowing movements. At this time the flow of urine which had been sparse and uncontrolled, increased, with incontinence at first, and a urinary antiseptic† was given by mouth. From this stage recovery was rapid and the dose of chlorpromazine was gradually reduced. By the 28th day the dog was able to walk into the surgery and medication was discontinued. There was a slight radial paralysis which did not persist.

Although there was some loss of condition this was not so marked as would be expected after almost 3 weeks of illness with a minimum intake of fluid and nourishment. When seen in December, 1958, the dog was active and normal in all respects except that there was a slight "risus sardonicus."

It must be emphasised that the recovery of this case was in very large measure due to the care and nursing of the owner. The effect of the drug in lowering metabolic activity appears to have minimised the loss of condition and it might be thought that a state akin to hibernation was maintained.

† Hexamine.

The Effect of Phenothiazine on Serum Albumen Levels and Erythrocyte Values of Nigerian Zebu Cattle

BY

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SUMMARY.—The effect of therapeutic and massive doses of phenothiazine on serum albumen and erythrocyte values in cattle is reported. It is suggested that the toxicity of phenothiazine is enhanced by poor condition or low nutritional levels in the bovine. Toxic doses of phenothiazine are shown to produce lowering of the serum albumen level. The mode of action of the drug and the likely rôle of the serum albumen drop in toxicity are discussed.

Introduction

THERE are numerous reports in the literature referring to the toxic effects of phenothiazine in cattle. Cauthen (1945) reports temporary blindness, anaemia, and death resulting from phenothiazine given at normal dosage rates to animals in poor condition. In New Zealand, Whitten, Clare, and Filmer (1946) cite keratitis in calves as a sequel to exposure to sunlight following phenothiazine medication. Bolton (1948) records prostration and death 24 hours after dosing at normal rates. A considerable variation in toxic effects on individual cattle is evident from these reports and from experiments where large doses of phenothiazine have been given. Cauthen (1945) gave a 260-lb. calf 250 g. of phenothiazine causing it to become unsteady on its feet 24 hours after dosing with a subsequent quick recovery. Other calves given up to 100 g. failed to show any toxic symptoms. Bruford and Fincham (1945) gave calves in poor condition 40 to 100 g. four times at fortnightly intervals without ill effect. Rogers, Brooks, and Higgs (1957) gave a 268-lb. calf 436.5 g. of phenothiazine in a single dose. Toxic symptoms were produced lasting up to 5 days and causing a loss of weight of 33 lb. No significant alteration in erythrocyte values nor any haemolysis were noted.

During a field investigation into poor thriving in calves in 1957 where parasitic gastro-enteritis was a major factor, it was noted that a group of calves receiving regular monthly therapeutic doses of phenothiazine had a mean serum albumen level of 1.78 g. per 100 ml. while a similar group receiving similar phenothiazine therapy but also receiving a protein supplement of ½ lb. of groundnut cake per day, had a mean serum albumen level of 2.84 g. per 100 ml. Another group, receiving mineral supplement only, had a serum albumen level of 2.3 g. per 100 ml. The comparable normal serum albumen level for cattle of a similar age group at Vom is 2.4 g. per 100 ml. (Peers, unpublished data). Subsequent to these findings 20 healthy Zebu calves at Vom, of a similar age, were divided into 2 groups of 10. One group served as a control while the other

TABLE I
COMPARISON OF CALVES W. & B. ERYTHROCYTE VALUES

Calf	Date	P.C.V.* per cent.	Hb. g./100 ml.	Rbc's 10^6 cu.mm.	M.C.H.C.† per cent.	M.C.V.‡ c/u
W	18.2.58	20·0	9·80	4·37	39·2	57·0
B	18.2.58	28·5	11·20	5·58	39·3	51·0
Vom Normal	—	35·9	11·40	10·44	31·7	34·3

* P.C.V.—Packed cell volume.
† M.C.H.C.—Mean corpuscular haemoglobin concentration.
‡ M.C.V.—Mean corpuscular volume.

received normal therapeutic doses of phenothiazine on each of the succeeding 3 months. No significant change in serum albumen levels or erythrocyte values were evident in the treated group. It was considered that condition and nutritional level were implicated in the previous results and further work was undertaken to interpret these findings.

Methods

Total serum nitrogen estimations were made by the micro-Kjeldahl digestion technique and the total serum protein in g. per 100 ml. was calculated by multiplying by a factor of 6.25. The serum albumen level was then calculated from the percentage albumen concentration estimated by paper electrophoresis of the sera, using the methods of Flynn and De Mayo (1951) and Jenks, Jetton, and Durrum (1955). Haemoglobin concentration was estimated using a Sahli haemoglobinometer and red cell counts with a Neubauer counting chamber. For packed cell volumes, oxalated blood was centrifuged at 3,500 r.p.m. for 1 hour.

Design of Experiments and Results

A. *Relationship of Phenothiazine to Dietary Protein*

Design. From December 27th, 1957, 2 calves were fed on a protein-deficient diet of Acha hay *ad lib.* only. On January 30th, 1958, calf W was dosed with 90 grammes of phenothiazine. It was subsequently dosed with 100 g. on February 10th, 1958, and 100 g. on February 17th, 1958. Calf B served as the control. Serum albumen and erythrocyte value estimates were made prior to each dose and 24 hours, 72 hours, and 120 hours after the third dose.

Results. A steady fall in serum albumen level was demonstrated in calf W as compared to a rise in calf B, the control. The fall was slight until February 17th and reached the lowest point on February 20th, 72 hours after the third dose of phenothiazine. Calf W also showed a normochromic macrocytic anaemia, and this was most marked 24 hours after the third dose.

The erythrocyte values of the 2 calves 24 hours after the third dose are shown in Table I, and the course of serum albumen values in Table II.

B. *Effect of Phenothiazine on Cattle with Low Serum Albumen Levels*

Design. Seven adult White Fulani Zebu bullocks were divided into 2 groups, 4 to be treated with phenothiazine and 3 to act as controls. These cattle had 3 to 4 weeks previously been used to test rinderpest dried goat virus vaccine and were recovering from a severe febrile reaction. The 4 bullocks were each given 50 g. of phenothiazine. Whole blood and sera were collected from the dosed bullocks and the controls prior to dosing, and 24 hours and 120 hours after dosing.

Results. Mean serum albumen levels and erythrocyte values for the two groups are shown in Table III.

The mean serum albumen value in the control group continued to rise throughout the trial period. In the phenothiazine dosed group there was a slight drop in the mean serum albumen value on the third day and evidence of recovery on the 6th day. There was also signs of a slight normochromic macrocytic anaemia in the dosed group, but a rise in the mean corpuscular cell volume on the third day in the control group makes this of doubtful significance.

C. *The Effect of a Lethal Dose of Phenothiazine on Erythrocyte Values and Serum Albumen Levels*

Design. Two White Fulani Zebu calves in poor condition were maintained for a few days on a protein-deficient diet of poor quality Acha hay. One animal, selected because it showed a relatively high

TABLE II
COMPARISON OF CALVES B. & W. SERUM ALBUMEN VALUES

Date	30th January	10th February	17th February	18th February	20th February	22nd February
Phenothiazine dosage to calf " W "	90 g.	100 g.	100 g.	—	—	—
Calf " W " serum albumen g./100 ml.	2·50	2·48	2·30	2·20	2·05	2·20
Calf " B " (control) Serum Albumen g./100 ml. ...	2·50	2·55	2·48	3·30	2·90	3·0

TABLE III
MEAN SERUM ALBUMEN LEVELS AND ERYTHROCYTE VALUES OF TEST AND CONTROL GROUPS OF CATTLE WITH LOW SERUM ALBUMEN LEVELS

Time sample taken	Group	Serum albumen g./100 ml.	P.C.V. per cent.	Hb. g./100 ml.	Rbc's 10^6 cu.mm.	M.C.H.C.	M.C.V. c/u
Prior to dosage	Phenothiazine	1·63	22·2	7·29	4·96	32·8	44·7
	Control	1·63	22·1	6·84	4·95	32·4	42·6
24 hours following dosage	Phenothiazine	1·31	23·3	7·01	4·71	30·0	49·4
	Control	1·75	23·6	7·46	5·06	31·6	46·6
120 hours following dosage	Phenothiazine	1·57	23·4	7·23	4·85	30·9	48·2
	Control	1·80	22·5	6·89	5·27	30·6	42·6

serum albumen, calf P, was then given 500 grammes followed by 2 doses of 300 and 700 g. two days later. The other calf, S, was not dosed and acted as a control.

Results. The first dose of 500 g. resulted in calf P displaying toxic symptoms. The animal was dull, unsteady on its feet, showed muscular tremors and pale mucous membranes. The condition of the animal improved within 24 hours though it remained dull. The final two doses totalling 1,000 g. produced violent symptoms. Initially, trembling of the leg muscles occurred accompanied by mild exophthalmia, and injected conjunctiva. Latterly the calf became recumbent when severe muscular spasms developed. These continued for a short while before death occurred. The main *post-mortem* findings were generalised petechial haemorrhages, slight icterus, and noticeable muscle flaccidity. This latter feature may possibly have resulted from capillary collapse and have been associated with the serum albumen drop described below.

Serum albumen levels and erythrocyte values for both calves over the dosing period are shown in Table IV. The serum albumen level in calf P dropped to 1.55 g. per 100 ml. following the 500-g. dose of phenothiazine, rose 24 hours later to 1.95 g., then fell to 1.33 g. just prior to death. It is interesting to record that the control animal S, which was in very poor condition, showed a serum albumen level of 1.37 g. per 100 ml. on the only day it was unable to rise. This fall, presumably a result of the poor condition produced by the low-protein diet, was, however, only 50 per cent. of the fall produced in the dosed animal P. The erythrocyte values increased following the heavy dosing. This haemoconcentration will be discussed later.

Discussion

From these investigations, 3 salient points arise concerning phenothiazine toxicity, namely, the alteration of erythrocyte values, the fall in serum albumen, and the susceptibility of animals on low-level nutrition or low-protein diet. The last 2 may be of considerable importance.

Alteration in erythrocyte values of the bovine following phenothiazine dosing is not a feature of previous reports on toxicity. Cauthen (1945) and Rogers *et al.* (1957) both failed to alter erythrocyte values significantly by heavy dosage. In horses, however, alterations in erythrocyte counts and packed cell volumes are frequently recorded. Swales *et al.* (1942) report lowered erythrocyte counts and haemoglobin values in horses and suggest that haemolysis had occurred rather than damage to the haemopoietic system. Our observations suggest that a macrocytic anaemia may develop following toxic doses and that this may be an effect of the lowered serum albumen levels producing lowering of the osmotic pressure of the plasma proteins which depends largely on the albumen molecules (Bard,

TABLE IV
SERUM ALBUMEN LEVELS AND ERYTHROCYTE VALUES OF CALVES S. AND P.

Calf	Date	Phenothiazine dosage	Serum albumen g./100 ml.	Hb. g./100 ml.	Rbc's 10^6 cu. mm.	P.C.V.	M.C.H.V.	M.C.V.
S	5.8.58	500 g.	1·64	7·18	5·05	21·0	34·1	41·5
P		500 g.	2·24	9·80	9·60	26·0	37·6	27·0
S	6.8.58	—	1·80	7·02	4·75	21·0	32·6	45·2
P			1·55	9·93	9·10	35·1	28·3	38·6
S	7.8.58	—	1·37	6·86	4·52	21·5	32·6	46·4
P		1,000 g.	1·95	13·44	12·0	41·0	32·7	34·1
S	8.8.58	—	1·80	6·44	4·30	22·5	29·3	50·0
			1·31	12·02	11·75	36·0	33·4	30·6

1941). The lowered osmotic pressure would produce a swelling of the erythrocytes and a macrocytic anaemia. As a macrocytic anaemia was not a feature in all the animals given phenothiazine and as considerable deviation in erythrocyte counts occur, the anaemia cannot be considered as significant.

The serum albumen fall would appear to be markedly affected by the condition of the animal and nutrition. In horses, Woolf and Simms (1943) failed to produce toxic effects with phenothiazine while they were on a high-protein diet. With horses fed on low-protein diet the same workers produced icterus, anaemia, and falls in packed cell volumes of 3 to 20 per cent. The findings on relationship to protein cited above, suggest that serum albumen levels following phenothiazine dosage may be affected by the level of protein in the diet. Instances in the field in Africa have been noted where Zebu cattle in poor condition have succumbed to normal doses of phenothiazine. It appears, therefore, that cattle in poor condition, or on low-protein level diets, may be considerably more susceptible to the toxic effects of phenothiazine.

The mode of action of the drug is interesting and may be explained by the work of Collier and Allen (1942) and Collier and Allenby (1952) who showed that phenothiazine inhibited the activity of succinoxidase in rat liver and beef heart due to the action of the sulphoxide derivative. Clare (1947) demonstrated that in calves, phenothiazine was absorbed from the intestine as the sulphoxide and was converted in the liver to phenothiazone. He found that the conversion of sulphoxide in the liver of cattle was not as efficient as in sheep and that some of the sulphoxide reached the peripheral circulation and aqueous humour. Clare *et al.* (1947) showed that the phenothiazine sulphoxide produced keratitis in cattle, when the animals were exposed to sunlight. As succinoxidase is associated with protein metabolism in the liver (Baldwin, 1952), the fall in serum albumen levels following phenothiazine may well be the result of toxic effects of the sulphoxide fraction on this enzyme and this would also account for the association with low dietary protein. Ellson and Todd (1957) demonstrated that no phenothiazine was left in the digestive tract of calves after 120 hours. This agrees with the results of the observations on cattle with low initial serum albumen levels, when the lowest of the 3 serum albumen levels was recorded 24 hours following dosing, and where evidence of recovery was seen 120 hours after dosing It would not be correct to assume from these results that the toxic effects of the phenothiazine are the result of the falls in serum albumen but it is feasible to postulate an association. Toxicity could either result from a rapid fall in the serum albumen over a short period, irrespective of the final level reached or from a drop to a final critical level. The latter we consider the more likely. A drastic lowering of the serum albumen would reduce the osmotic pressure to such an extent that fatal shock would ensue. Haemo-concentration, which occurred in calf P, at the same time as a lowering of the serum albumen is stated to be the earliest detectable sign of threatened circulatory deficiency of capillary origin (Gradwohl, 1948). Fatal shock is known to occur in humans when the plasma proteins are reduced to under 1 per cent. (Thorpe, 1949). We have been unable to find similar recorded data for cattle but our hypothesis would suggest that a considerably higher percentage plasma protein level is critical and that in Zebu cattle the critical level for serum albumen is around 1.3 g. per 100 ml., when evaluated by electrophoretic analysis. It seems not unlikely, therefore, that a lowered serum albumen level is an important factor in the toxic action of phenothiazine.

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References

BALDWIN, E. (1952). "Dynamic Aspects of Biochemistry, the Citric Acid Cycle." Cambridge University Press. Pp. 436-8.

BARD, P. (1941). "McLeod's Physiology in Modern Medicine: Osmotic Pressure of Plasma Proteins." H. Kimpton, London. Pp. 447-8.

BOLTON, J. (1948). *Vet. Rec.* **60.** 479.

BRUFORD, J. W., & FINCHAM, I. H. (1945). *Ibid.* **57.** 421-4.

CAUTHEN, G. E. (1945). *J. Am. Vet. Med. Ass.* **112.** 71-2.

CLARE, N. T. (1947). *Aust. Vet. J.* **23.** 340.

———, WHITTEN, L. K., & FILMER, D. B. (1947). *Ibid.* **23.** 344.

COLLIER, H. B., & ALLEN, D. E. (1942). *Canad. J. Res.* **20.** 284-90.

———, & ALLENBY, G. M. (1952). *Canad. J. Med. Sc.* **30.** 443-6.

ELLSON, T., & TODD, A. (1957). *Am. J. Vet. Res.* **68.** 519-29.

FLYNN, F. V., & DE MAYO, P. (1951). *Lancet.* **2.** 235.

FINCHER, M. G., & GIBBSON, W. J. (1941). *Cornell Vet.* **31.** 220-3.

GRADWOHL, R. B. H. (1948). "Clinical Laboratory Methods and Diagnosis." Volume I. H. Kimpton, London. Pp. 958.

JENKS, W. P., JETTON, M. R., & DURRUM, E. L. (1955). *Biochem. J.* **60.** 205-15.

PEERS, F. G. (1958). White Fulani Blood Values. (Unpublished data.)

ROGERS, R. L., BROOKS, F. W., & HIGGS, B. G. (1957). *Vet. Med.* **52** (5). 219-24.

SCHMIDT, A., CHRISTIAN, T., & SMOTHERMAN, W. (1941). *J. Am. Vet. Med. Ass.* **99.** 225-9.

SWALES, W. E., COLLIER, H. B., & ALLEN, D. (1942). *Canad. J. Res.* **20.** 349-61.

THORPE, W. V. (1949). "Biochemistry for Medical Students: Functions of Plasma Proteins." J. A. Churchill, Limited. 159-61.

WHITTEN, L. K., CLARE, N. F., & FILMER, D. G. (1946). *Nature, London.* **157.** 232.

WOOLF, F. P., & SIMMS, B. T. (1943). *N. Am. Vet.* **24.** 595-9.

The "Hamama" Case

BY

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SPECIAL NOTE

The following greatly condensea account has been compiled partly from various reports in the national press, and partly from facts and information available to the Equine Research Station of the Animal Health Trust. It is well known that when technical evidence is given before a court of justice, reports prepared by lay journalists tend to be compressed, and sometimes emphasis is given to less important, but perhaps more dramatic, aspects, while important technical evidence is omitted, misunderstood, or may be unintentionally distorted. Consequently, it has been thought advisable to reinforce, wherever possible, the accounts which have appeared in various newspapers by other available reports and information, so that our readers may obtain a more comprehensive appreciation of the facts of this case.

Chronological Record of Events

ON the fine, warm afternoon of May 2nd, 1956, the Two Thousand Guineas Stakes, the first classic race of the year for three-year-olds, run over one mile, was won by the 50-to-1 outsider "Gilles de Retz." Another outsider, at the same starting price, finished a bad last in a manner described as "swaying like a drunken man." This was the French colt "Hamama," by "Relic" out of "Haldis," which only 10 days before the race, had been sold by Prince Said Tousoun and Mme. Cunnington, through the St. Simon Bloodstock Agency, to Mr. John Lewis, former Labour M.P. for Bolton West, for £7,000 with a contingency of £5,000 if he won the "Two Thousand."

Mr. Lewis, disappointed by "Hamama's" performance, immediately stopped his cheque to the Agency on the grounds of a breach of warranty. Eventually, some 6 months later, on November 9th, 1956, "Hamama" was destroyed, on Mr. Lewis's instructions, at the Equine Research Station of the Animal Health Trust. As a result, Prince Tousoun and Mme. Cunnington sued the Agency and Mr. Lewis for £7,000.

In another action the Agency sued Mr. Lewis for £7,420 which included commission, etc. Mr. Lewis counter-claimed for damages.

The case was heard by Mr. Justice Paull in the Queen's Bench Division of the High Court. It began on March 2nd, and lasted for 9 days. At the end of the hearing Mr. Justice Paull reserved judgment, and admitted that it "caused him a great deal of trouble." Eventually, on March 20th, 1959, he gave his Judgment (which will be published verbatim next week) in favour of the defendant.

History of "Hamama's" Career

For convenience, the events have been recorded in chronological order, but it is first necessary to consider "Hamama's" history before he started in the fateful classic on May 2nd.

He was foaled in the spring of 1953 and, owing to what Mr. Beyfus Q.C., for the Plaintiffs, referred to as "a defect in his pedigree which made him unsuitable for stud purposes," was given by Prince Tousoun to his trainer, Mr. J. E. Cunnington, in July, 1955. He was described as a backward colt and was not raced until October, 1955. Then, however, in a period of 16 days he ran 3 times, finishing third in his first race, fifth a week later, and winning his third race, beating "Lavandin" who, 8 months later, won the Derby. On April 15th, 1956, in his first race as a three-year-old, he finished second in a tough contest in heavy going. Thereafter, before being flown to England on April 23rd, he was given no fast work. On that date he entered the stables of Sir Gordon Richards, Mr. Lewis's trainer, who told the court that, after consulting Mr. Cunnington, he had merely given "Hamama" walks, canters, and half-speed work until April 30th, when he gave him his final canter over 5 furlongs, and a half-speed gallop for 5½ furlongs. The colt left Sir Gordon's stables at Ogbourne on the morning before the race with his "boy" and the travelling head lad, and spent the night in the Jockey Club stables at Newmarket, where strict security precautions were enforced, as usual. On the morning of May 2nd, he had an hour's "loosening" exercise.

As to what occurred in the race, there seems to be little or no disagreement, so it may suffice to refer to the evidence of "Hamama's" jockey, Rickaby, who testified that the horse went quite satisfactorily for about 3 furlongs. When, however, Rickaby tried to improve his position, "Hamama" rolled first to the left and then violently to the right, later staggering to such an extent that the jockey thought he would fall. He was eased up and cantered past the post, "tailed-off," and walked to the paddock. Rickaby added that after the first swerve the horse made a noise which was harsh rather than a whistle —a point which will be referred to later.

Sir Gordon Richards said that after the race "Hamama" was staggering like a drunken man and very distressed. He therefore sought, at once, the assistance of Mr. F. T. Day, one of the official Jockey Club veterinary surgeons, who examined the horse immediately, and again about 6 p.m., by which time he had cooled off and regained control of his movements but was still "like a drunken man."

Veterinary Examinations of "Hamama"

Mr. Day, in his evidence given on the fifth day of the case, agreed that after the race he found "Hamama" in great distress with his heart in a state of palpitation, "going at a terrific rate." The lungs were normal but breathing was very rapid and movements were incoordinated.

"Hamama" was admitted to the Equine Research Station, Newmarket, on May 3rd by arrangement between Mr. Day and the Director of the Station, who, on the sixth day, told the court that the horse, after a very comprehensive examination, was found to have a dilated heart, and a heavy infestation with parasitic worms, the count being 3,200 eggs per gramme. There was marked variation of heart rhythm, and of the audibility of the first sound in each heart beat. After 20 minutes' lungeing the stethoscope revealed acceleration of the heart beat, and a systolic murmur could be heard following the first sound. Electro-cardiographs showed that the rhythm of the heart was irregular, there were partial heart blocks and supplementary S waves were present. "We were sure that the horse's heart had been strained and that the consequent dilatation had not returned to normal." "Hamama" left the Equine Research Station on May 8th, 1956, to be given strictly graduated exercise in the Newmarket stables of Mr. R. A. Jones, prior to re-examination in the autumn.

On May 15th, the horse was examined by Major A. C. Foster, M.R.C.V.S., who stated in his certificate that when the horse was at rest in the stable his heart beats were irregular, and after exercise they became very irregular, while later he detected a slight murmur. On May 16th he examined the horse again and found the same state of affairs. He found it was "impossible to believe that 'Hamama' was not suffering from cardiac irregularities at the time of his export from France. An examination at the time of purchase should have revealed this."

On June 4th, an independent examination of "Hamama" was carried out by Mr. F. W. Chamberlain, M.R C.V.S., who later gave evidence to the effect that he had found a slight intermittence of heart beats at regular intervals, which, in his opinion, rather surprisingly disappeared after a good gallop.

M. André Bouchet, a French veterinary surgeon, who saw "Hamama" at the same time, testified that the only defect was an irregular intermittence of the heart, not unusual in race-horses and not serious. It disappeared with work.

On October 16th, the horse was readmitted to the Equine Research Station for re-examination which, according to Mr. Miller's testimony, disclosed evidence of heart blocks, as indicated by arrested heart beats, valvular incompetence, and an unusual post-systolic murmur developed with vigorous exercise, which disappeared as the heart slowed down subsequently. The chances that "Hamama" might ever be fit for racing were not good—perhaps 50-50.

On October 20th Mr. F. W. Chamberlain again examined "Hamama" at the Equine Research Station and stated in his report that the horse looked well, but was lighter in condition. He gave his opinion that in the hands of a capable trainer the horse might rehabilitate himself by the spring, and added that there was not the slightest justification for slaughter either on humanitarian or economic grounds. To that opinion he adhered in his evidence at the hearing.

Under instructions from Mr. Lewis "Hamama" was destroyed on November 9th, 1956. An autopsy was carried out by Mr. L. W. Mahaffey, at the Equine Research Station, in the presence of Dr. Bouchet, Mr. Chamberlain, Lt.-Col. Hickman, Major Townsend, and Mr. Miller.

This, said Mr. Miller in evidence, revealed fenestrations in the heart which would have materially affected its action. They could account for the unusual post-systolic murmurs previously mentioned. "Hamama's" behaviour during the race was consistent with what would have been expected to happen if the knowledge of the valvular incompetence, dilated heart, and the fenestrations had been available before the race.

Regarding this *post mortem*, Mr. Chamberlain testified that, when the pathologist of the Equine Research Station opened the heart, there was a "slight tear" in one of the valves. He had also been shown electro-cardiographs of the horse's heart but he was sceptical of their practical value. He also expressed the view that the mere act of submitting an excitable race-horse to electro-cardiographs might cause a series of fluctuations in the picture. M. André Bouchet testified that the *post-mortem* examination showed nothing abnormal in the heart.

This, then, was the sequence of events, and now comes the obvious question, "What about veterinary examination and certificate prior to purchase?"

It can be said at once that, despite the large sum of money involved, no veterinary certificate of soundness was supplied either to Mr. Lewis or to Sir Gordon Richards, nor was one produced in court. In cross examination, Mr. Cunnington admitted to Mr. Milmo, for the Agency, that he had told Sir Gordon that "Hamama" was a good and sound horse. Opening the case for the plaintiffs, Mr. Beyfus said that before the horse left France he was found by a veterinary surgeon to be perfectly fit and sound in wind and limb, and to have a sound heart.

M. André Bouchet, the French veterinary surgeon previously mentioned, affirmed that he gave "Hamama" a thorough examination before he was sent to England; his general condition was good and his heart perfectly normal. He had auscultated the heart for one minute at rest, and again for a similar time after exercise. He had not been asked to furnish a veterinary certificate of soundness for "Hamama." Sir Gordon Richards fully realised the importance of a veterinary certificate. His testimony as to his conversation over the telephone with Captain J. de Moraville of the St. Simon Bloodstock Agency, as reported in the *Daily Telegraph*, is worth quoting in full, for it constitutes a serious warning to those who are apt to regard certificates of soundness too lightly. The quotation is as follows: "'I said to him: "You know if this deal does

go through it is subject to a veterinary certificate." He replied: "You know, Gordon, you need not worry about that at all. I have already had him vetted. I have a veterinary certificate and I warrant him sound." ' Sir Gordon said he then telephoned Mr. Lewis. He again called Captain de Moraville and said: 'All right. It's a deal. Naturally, John, you have bought French horses before: be sure about this warranty.' After the race, in which the horse finished last, Sir Gordon said he telephoned Captain de Moraville. 'I said: "Well, John, that horse ran badly to-day," and asked him what he was going to do about it. I told him that the veterinary surgeon had found that the horse had "got a heart" and not only that, the jockey had told me the horse made a noise. He said: "Good God. What is going to happen now?" I said we must wait and see.' "

It may now be of interest to consider the possible causes of "Hamama's" failure in the "Two Thousand" as suggested by various witnesses. Asked what he thought was the most likely event in "Hamama's" life to have caused his breakdown in the "Guineas," Mr. Cunnington, a trainer of some 37 years' standing, said that there was a complete change of air temperature at that time. Pressed on the point he said that might have been a factor, but possibly not the real factor. Further questioned, he said he did not know the most probable cause but there was the possibility of the horse having been interfered with before the "Guineas."

At this point it should be made quite clear that although there was a good deal of reference throughout the hearing to the possibility of "Hamama" having been doped, Mr. Beyfus, for the plaintiffs, said that he was not suggesting that anything of that sort could have happened with the knowledge of Mr. Lewis or Sir Gordon Richards.

The Question of Possible Doping

M. André Bouchet could think of no explanation other than a strained heart, unless the animal had been interfered with. If that had occurred, it must have been before going into the parade prior to the race—the symptoms would show within 10 to 20 minutes of the dose being given.

Mr. F. W. Chamberlain, in answer to Mr. Beyfus's question as to what would cause a horse to behave in the way "Hamama" did, replied, "I must say the administration of a pernicious drug. It is a possibility that one would like eliminated." An alkaloid—a stimulant—was the sort of drug he had in mind. He believed cocaine and caffein were the old stand-bys and, more recently, benzedrine. He told the Judge that it was difficult to know how long it would be before the first symptoms of doping showed, and how long the effect would last.

Asked what amount of a drug would be required to produce symptoms, Mr. Chamberlain said it would vary. He remembered one case in which an animal had received 16 grains of cocaine and he thought the symptoms manifested in this case would be compatible with a dose of that amount. It would be necessary to experiment to find the right dose for a horse, and he agreed that the 7 days during which "Hamama" was in Sir Gordon's stable did not afford much time for experimentation. The symptoms of an overdose of cocaine, he said, would be great excitement, nervous disturbance, incoordination of movement, reeling, sweating, staring eyes, and in extreme instances, even delirium. Such symptoms would lead him to suspect overdoping and he would expect a saliva and sweat test to be made. The Judge said he assumed Mr. Day examined "Hamama" thoroughly and did not find these symptoms—would Mr. Chamberlain then rule out drugs? Mr. Chamberlain said he was impressed by Mr. Day's report. The horse was distressed and showed loss of coordination of all limbs, and was swaying about in a drunken manner. Those were symptoms, and he would have liked the possibility of overdoping excluded by examination of sweat and saliva.

Sir Gordon Richards stated that although there was something very, very wrong with "Hamama's" performance in the race, he had no thoughts of drugs or doping and he saw nothing after the race to make him suspect that the horse had been doped.

Mr. F. T. Day, on cross-examination, said that he found nothing in his examination on May 2nd which primarily, on its own, suggested dope, but he could not exclude the possibility.

Mr. Wm. C. Miller, M.R.C.V.S., F.R.S.E., in his evidence which took up the whole of the sixth day, and the first half of the seventh, told Mr. Lawton, Q.C., for Mr. Lewis, that in his opinion, none of the evidence was consistent with "Hamama" having been doped before the Two Thousand Guineas. The signs of doping would have been manifest before the "off" unless the timing of the dope had been so precise that the drug only took effect when the race was actually in progress, which he considered most unlikely. He thought that the horse's performance was consistent with the occurrence of the second of two strains of the heart. The most likely explanation was that the first strain occurred during the race on April 15th, less than 3 weeks before the Newmarket race. Everything pointed to a previous strain of the heart which had not been noticed at the time.

Probable Causes of Poor Performance

To the Judge's question, "Do you think this is a probable cause, or a possible cause?" Mr. Miller replied, "I think, taking everything into consideration, it is the most probable cause." In answer to a question from Mr. Beyfus, Mr. Miller pointed out that "Hamama" "faded out" early in the "Guineas" and said, "A 'fade out' towards the end of a race, when a horse is asked for an extra effort, is typical of strain and dilatation occurring for the first time. It is the extra effort when the horse is under pressure in the last furlong, or so, which does the damage."

Major A. C. Foster gave evidence in support of the certificate which he had given after examining "Hamama" 12 days after the race. (See above.)

Closing Speeches

Mr. Lawton submitted, in his closing speech for the defence, that clearly on the evidence there were

no grounds for suggesting that "Hamama" might have been doped.

The Judge said he had been wondering about the position of a horse that had got a permanently dilated heart, but of such a character that it could not be discovered by an ordinary veterinary examination with a stethoscope. Was the horse sound or unsound when sold? Mr. Lawton replied that he would have said unsound. It was for the seller, who was guaranteeing the horse sound, and selling him as a race-horse fit to run in the Two Thousand Guineas, to show that he was sound. His Lordship said he was not sure whether a warranty of a horse was exactly the same as soundness of a motor car that had a latent defect that could not be discovered at the time of sale and, later, caused a breakdown.

Mr. Beyfus, winding up for the plaintiffs, submitted that on the evidence one was left in the state of having to guess as to the condition of "Hamama," as distinct from a proper reasonable inference of facts. The Judge said he felt Mr. Beyfus would have a very strong case indeed if the horse had broken down in the last furlong or so, and he would have had very little hesitation in finding for him. The difficulty he felt about it was the breakdown in such an early stage of the race. It did look as though something was wrong with the horse's heart before the race started.

With that the hearing virtually ended, His Lordship reserving judgment.

Other Points in the Case—Whistling

The question of "Hamama" being a whistler has already been noted in connexion with Sir Gordon Richard's account of his telephone conversation with Captain de Moraville after the race. This condition was included by Mr. Lewis in his complaint of breach of warranty, and, as already recorded, the jockey Rickaby testified that "Hamama," after his first swerve, "made a noise" which decreased when he eased the horse and increased when he attempted to accelerate. It was, however, a harsh noise, a little different from a whistle. The evidence for the horse having made a whistling noise was first contained in a certificate given by Mr. Day. He had had the horse galloped at half speed for four furlongs and full out for a further furlong on May 23rd. As the horse passed him he quite clearly heard a whistle. This was fully confirmed by Mr. George Molyneux, who rode the horse in the graduated exercise given to him after he left the Equine Research Station. He was quite definite that "Hamama" was a whistler. "You could hear it every time you asked him to go faster." Once when Mr. R. A. Jones (the trainer in charge of the horse at this period) was riding "Hamama" past him, 30 to 40 feet away, he (Molyneux) could still hear it.

Mr. Cunnington in evidence said that if "Hamama" had been a whistler by April 15th, 1956, he did not think it would have escaped his notice. The horse was absolutely sound in wind.

Sir Gordon Richards apparently never heard the horse whistle and said, in his evidence, that if whistling had been heard while "Hamama" was in his stables, it would have been reported to him. Asked what the horse's value would have been if he had won the "Two Thousand" despite being a whistler, Sir Gordon caused considerable amusement by his reply, "If a whistler started overnight, he could not win the 'Guineas.'" He agreed, however, that a horse with an incipient whistle might win.

The Reasons for Destruction

Another point which has provoked considerable discussion is the action of Mr. Lewis in ordering the destruction of the horse. His explanation to the Judge was as follows:—

"After the horse had gone to the Equine Research Station, and I had received the original reports from them, from Mr. Fred Day and Major Carey Foster, and after the horse had had 6 months of graduated exercises in the yard of Mr. R. A. Jones, the horse then went back to the Equine Research Station for further examination. The information I received at the time from the Equine Research Station was that they were disappointed in the progress that had been made in the horse's heart, which was still very bad. In the circumstances, as 'Hamama' was a three-year-old then, and the next season would have been a four-year-old, and the value of a four-year-old which has not won a race as a three-year-old, and where there is the slightest suspicion about any heart or wind condition, is very low—after consultation with my veterinary advisers, I decided to give instructions to the Equine Research Station to put the horse down. Indeed, I think it was the humanitarian thing to do."

Answering Mr. Beyfus, Mr. Lewis said he offered the horse back to Prince Tousoun and Mr. Cunnington for £7,000, which was their price, assuming "Hamama" to be a good, sound, horse. He did not offer "Hamama" at auction because its value would have been practically negligible. When Mr Beyfus observed "I understood the object of putting the horse up for sale was to fix the damages," Mr. Lewis replied that that was what he was advised to do, but he had also been assured that he would not have got a bid for the horse in that condition. Another reason for destruction was that the specialists at the Equine Research Station had said that it was impossible to determine precisely the presence of a lesion in the heart without a *post-mortem* examination. Mr. Beyfus then pointed out that Mr. Lewis had heard Mr. Miller say that he would have kept the horse alive if it had been left to him. Mr. Lewis's reply was to the effect "they" (presumably the staff of the Equine Research Station) "always like to keep horses alive there because they don't have to pay for them. They cost £10 to £15 a week to keep."

Summary of Judgment

On March 20th, Mr. Justice Paull gave judgment in favour of Mr. Lewis for £7,000, the sale price of "Hamama."

In the first action, His Lordship entered judgment for the plaintiffs for £405 against Mr. Lewis, and for Mr. Lewis for £630 on his counterclaim, leaving a

(*Continued at foot of col.* 1, *page* 485)

Current Literature

BOOK REVIEWS

The Metabolism of Sulphur Compounds. By L. YOUNG and G. A. MAW. 180 pp. with numerous references. 1958. Methuen & Co. Ltd., London. 16s.

This is a good and timely addition to the excellent series of monographs on biological subjects and is interesting from several points of view. In the first place it is an admirable illustration of the ways in which scientific fashions change, for a few years ago phosphorus seemed to dominate the scene, but now, as this monograph indicates, sulphur is beginning to show up very prominently. Secondly, the book does achieve its purpose in presenting a lucid account of reactions occurring in living organisms in which sulphur plays a necessary part: indeed, in a variety of such reactions it is made clear that sulphur probably takes precedence of phosphorus. Lastly, it shows how nature anticipated some of the earlier and, one would have guessed, improbably "biological" excursions of the organic chemists of the 19th century: the account given of the metabolism of the sulphonic acids in Chapter 5 is a good example.

The introductory chapter gives a brief summary of the types of sulphur compounds found in living organisms and describes the methods which may be used for their study. In subsequent chapters the various types of compound are considered in detail, as follows: thiols and disulphides (2); thioethers (3); sulphonium compounds (4); sulphuric acid esters (6); miscellaneous organic compounds of sulphur (7); and inorganic forms of sulphur (8). The last chapter discusses the microbiology of sulphur.

Such a list of titles makes the volume sound "organic," rather than biological: this is far from the case. When one remembers that there is penicillin amongst the antibiotics, thiamine and biotin of the vitamin group, coenzyme A among the important enzymes, and methionine in the list of essential amino-acids, all of which contain sulphur, quite apart from the elimination of certain toxic substances from the animal body in the form of sulphur derivatives, one must realise beyond any doubt how prominent a biological rôle this element plays. Moreover, this list is by no means exhaustive.

The text is clear and concise, whilst the references, some 900 in number, include a very large proportion of recent ones, since the field has been a very active one during the last decade: the book is well printed. Probably the veterinary-minded reader will wonder why wool seems to have no mention, nor yet the complicated inter-relationship of inorganic sulphate, copper, and molybdenum. These minor criticisms apart, the book can be strongly recommended.

Canine Medicine. 41 authors. 2nd edition (revised). 860 pages. 296 illustrations. 1959. American Veterinary Publications Inc., Santa Barbara, California. 144s.

The second and revised edition of "Canine Medicine" appears 6 years after the first, a reprinting having been necessary in the interval.

The arrangement and style of the book is unchanged but 3 new authors make their bow although only 2 chapters are new to this edition. The text is increased by some 200-odd pages, in part due to the additional chapters on fluid therapy and paediatrics, in part to a greatly expanded (by some 50 pages) section on the heart, and also by additions to the text in many other chapters.

Parenteral fluid therapy is discussed briefly and in an interesting manner—probably the most useful point for the small-animal clinician is the inclusion of a formula to calculate the dose of whole blood required to raise the haemoglobin by 1 per cent. Elsewhere in the book the veterinary surgeon will learn, doubtless with some surprise, of the amount of blood which may safely be taken from a donor. In this country, where the keeping of canine blood donors as such is rare, there may be some justifiable reservations before subjecting "volunteers" belonging to one's clients and even one's own pets to such extensive bleeding.

Paediatrics now enjoys a chapter to itself, but the fact that much of the material is dealt with in other existing chapters somewhat detracts from its value. This section suffers from a tendency to vagueness, pregnancy diagnosis in the bitch being said to be readily made between 28 and 35 days, whereas by the latter stage in many cases foetal units are no longer palpable. Surely the statement that puppies start to shed their deciduous teeth between 2 and 4 months of age could have been far more precise? The reviewer can only presume that the recommendation to feed newly born orphan puppies at 8-hourly intervals is a misprint. As in many other places this chapter displays curious anomalies in emphasis, rickets being dismissed in a few lines while the comparatively uncommon hypertrophic osteodystrophy apparently merits several times that amount of detail.

It is indeed pleasant to be able to give the highest praise to the greatly expanded section on the heart. Detweiler is a real master of his subject and the

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The "Hamama" Case—*Concluded.*

balance of £225 in Mr. Lewis's favour. Plaintiffs were ordered to pay three-quarters of Mr. Lewis's costs. The amount of £405 was arrived at by assessing "Hamama's" value at the July Sales, when he might have been sold, at £525. Against this, the Judge put the cost of his keep, for twelve weeks, at £120. The £630 awarded to Mr. Lewis on his claim for special damages included commission payable to the Agency, insurance, veterinary fees, etc. In the second action, judgment was entered for the St. Simon Bloodstock Agency for £420, with no order as to costs.

Leave to appeal was granted in both actions.

(A verbatim report of the Judgment, except for the purely legal argument, will be published next week.)

reader has the advantage of a virtual monograph covering every aspect of canine cardiology.

In our previous review we were constrained to make several criticisms and it is with regret that we note that, despite the revision, the ground for such criticism in many instances persists. There is still far too much material of a purely surgical nature, clinical descriptions lack precision, and recommendations of lines of therapy and prophylaxis are still left very vague. The disproportion in emphasis as between common (and thus important) and the more rare conditions still exists.

Several authors deplore the absence of literature on certain diseases they describe yet the British literature has in fact supplied this in the period under review, a pungent comment on the all too familiar parochial treatment of literature which in most cases virtually ignores anything not North American in origin.

"Canine Medicine" remains a very desirable possession for the small-animal practitioner and is still the only book devoted to medical conditions of the dog, yet, although containing a tremendous amount of valuable information, it continues to fall just short of the promise it holds out to readers and still requires to be studied with critical care by those who already have a sound foundation of clinical experience.

Topographical Anatomy of the Dog. By O. C. BRADLEY, revised by TOM GRAHAME. 6th edition. 1959. Oliver & Boyd, Edinburgh and London. 36s.

Apart from certain new material the contents of this revised edition differs in no way from that of its predecessor, the 5th edition, published 9 years ago, the merits of which are too well known to require reiteration. The additions amount of 12 pages of text, 6 plates, and 4 diagrams. They include fresh material relating to the brain, autonomic nervous system, cardiovascular system, adrenal and pituitary glands, and the testicles.

It is particularly refreshing to find interest focussed anew upon structures of such practical importance as the ovarian artery, the course of which is now described in some detail. That such inaccurate phraseology as "The (carotid) sinus is formed by a number of sensory nerve endings . . ." (p. 201) has been allowed to creep into a book such as this is, however, disappointing.

Radiology is of such great practical importance in modern veterinary practice that the decision to include in this revised edition 6 completely new radiographs deserves nothing but praise. It is all the more regrettable, therefore, that, like those reprinted from the previous edition, the new radiographs are not of uniformly high standard. Indeed it is difficult to understand why such uninformative films as Plate VII (old) of the thorax, and Plate XVIII (new) of the mandibular articulation should have been allowed to appear in company with such excellent reproductions as Plate X (new), a renal pyelogram. Plate XIII (old) shows radiographs of the lateral and of the medial aspects of the tarsal joints. The very nature of X-rays renders the difference between these 2 radiographs negligible, and one might profitably have been replaced by an anteroposterior film of the joint with individual tarsal bones identified. Plate XX (new), a radiograph of the cysterna magna, is also open to criticism in that it might well mislead a student into supposing the cysterna to be situated entirely within the atlas.

The 4 new diagrams relate to the distribution of the sympathetic and parasympathetic divisions of the autonomic nervous system, to the ciliary body and iris of the eye, and to the subarachnoid space. All match the high standard set by the remainder of the 132 diagrams in the book.

As Professor Grahame has clearly emphasised in his preface, this book is intended to be a dissection guide, not a comprehensive text-book of canine anatomy. As such it will doubtless continue to be valued highly for many years to come and at the modest price of 36s. is excellent value for money.

Clinical Veterinary Surgery. By R. H. SMYTHE. Vol. 1. (1959). Crosby Lockwood, London. Pp. 355. 42s.

A new text-book on the subject of veterinary surgery and diagnosis is always welcome, especially when the author is a man of acknowledged experience and ability. "Clinical Veterinary Surgery," by Mr. R. H. Smythe, is introduced by a chapter dealing with the general principles of surgery and surgical pathology followed by chapters devoted to the external surface of the body, the eye, neoplasms, fractures, lameness, and the alimentary, urinary, and genital tracts.

These contain much useful information and advice, particularly the chapters on lameness and the examination of the abdomen, but suffer from lack of method in the presentation of the facts. Amongst the omissions noticed, a hernia is not distinguished from a rupture, specific names quoted in the text are not always supported by references in the bibliography, and any book purporting to deal with diagnosis can hardly afford to omit comprehensive guidance to its readers on differential diagnosis.

The author's style of writing is unusual. In an attempt to simplify complex physiological processes he is in danger of underestimating the intelligence of his readers, exemplified in this description of the clotting of blood. "Blood, as it flows from a small artery, is at first fluid. As it impinges on the torn ends of the vessels . . . it attains the consistency of a thin jelly."

The book is illustrated throughout by line drawings, and by a series of 16 photographic plates, many of which tend to illustrate the grotesque rather than the commonplace.

The ever-increasing output of scientific literature continually adds to our understanding of disease processes, their diagnosis, and treatment, so that the time has passed when a book of 355 pages can claim authority in so wide a field. It is hoped that the promised second volume will aim to cover less ground more thoroughly, and thus offer its readers greater stimulation.

BOOKS RECEIVED

Practical Animal Husbandry, by Miller & Robertson, 7th edition, 1959. Oliver & Boyd Ltd., Edinburgh and London. 31s. 6d.

Son of the Soil, by G. G. Rennie. Aberdeen University Press Ltd. 14s. 6d.

A Study of Benign Enzootic Paresis of Pigs in Denmark, by Aage Thordal-Christensen. Carl Fr. Mortensen Ltd., Copenhagen. 35s.

Implantation of Ova—Memoirs of the Society for Endocrinology No. 6, edited by P. Eckstein, 1959. Cambridge University Press, London. 30s.

Diet in Relation to Reproduction and the Viability of the Young, Part II—Sheep: World Survey of Reproduction and Review of Feeding Experiments. Commonwealth Bureau of Animal Nutrition, Technical Communication No. 20, by W. Thomson and F. C. Aitken, 1959. Co. Ag. Bureaux, Bucks. 30s.

Grass Productivity, by Andre Voisin, 1959. Crosby Lockwood & Son Ltd., London. 50s.

Silage and Haymaking, by Ian Moore, 1959. Farmer & Stock-breeder Publications Ltd., London 9s. 6d.

Mares, Foals and Foaling, by Friedrich Andrist. J. A. Allen & Co., London. 5s. 6d.

Reproduction in Domestic Animals, by H. H. Cole & P. T. Cupps, 1959. Academic Press Inc., New York and London. $14.50.

Principles of Humane Experimental Technique, by W. M. S. Russell and R. L. Burch, 1959. Methuen & Co. Ltd., London. 30s.

ABSTRACTS

Systemic Activity of Sevin in Control of *Ornithonyssus sylviarum*. KRAEMER, PAUL, & FURMAN, DEANE P. (1959). *J: Econ. Entom.* **52.** 170.

In a series of small-scale tests Sevin, N-methyl-l-naphthyl carbamate, eradicated Northern fowl mite infestations from laying hens when included in the feed at the rate of 3,000 p.p.m. Birds remained free of mites during a 21-day post-treatment observation period. Although birds received food containing Sevin for periods up to 8 days without showing any toxic reaction, medication for one day was sufficient to eradicate mites. No mortality or ill-effects were observed amongst specimens of *Cimex columbarius* which were allowed to feed on treated birds, suggesting that the systemic activity of Sevin may be selective in nature. J. C. W.

Toxoplasmosis in Sheep in England. RAWAL, B. D. (1959). *Lancet*. April 25th, 1959. Pp. 881-2.

Toxoplasma is a protozoon widely distributed among animals, including man, throughout the world. As a rule it behaves as a well-adapted harmless parasite; only rarely does it produce disease. In the human species it may cause severe congenital brain damage, leading to death before or after birth, or to physical and mental impairment. Less infrequently it is responsible for generalised glandular enlargement, mostly in children and adolescents. The question of the source of human infection is unsolved. Work in the U.S.A. suggested the possibility that man is infected by eating infected meat. This is borne out by the isolation of toxoplasma from 11 out of 50 pig diaphragms examined. In the Netherlands toxoplasma were isolated from 4 out of 30 sheep brains examined.

This paper describes first a preliminary survey of sera obtained from 100 pigs and 100 sheep slaughtered at the Sheffield abattoir. Reactions at a titre of 1 : 64 were found in 3 samples of sheep serum but in none of the samples of pigs' serum.

Blood samples and brains were obtained from a further 100 sheep. The brains were stored at 4° C. until the results of the tests in sera were known. Out of the 100 samples of sera 21 reacted to a titre of 1:4 or more. The brains of these 21 sheep were then examined, 11 produced neither antibodies nor cysts in inoculated mice, 4 caused only low-titre antibodies to appear. The remaining 6 sheep brains were responsible for the appearance of antibodies and cysts in inoculated mice.

From these observations it is concluded that sheep in England are affected with toxoplasma and that human infection might arise from the consumption of mutton—only, however, if it were eaten raw, for toxoplasma cysts are killed by 10 minutes' exposure to a temperature of 60° C.

G. F. B.

Epidemiology of Toxoplasmosis. ANON. *Lancet*. April 25th, 1959. Editorial, Pp. 869-70.

It addition to its wide geographical distribution toxoplasma has an amazingly wide host range. It seems to be capable of infecting all species of mammals, many birds, and some reptiles but not amphibians. The incidence in various species of mammals is quite considerable. In man, of those tested, only the Eskimos of Alaska are apparently free. Despite the vast amount of infection toxoplasma rarely does harm. The foetus is particularly susceptible and congenital infection is well known. Usually infection after infancy passes unrecognised. Sometimes it causes lymphadenopathy and 5 to 7 per cent. of cases of "glandular fever" are said to be reactions due to toxoplasmosis. Rarer manifestations are myocarditis, encephalitis and a severe and often fatal typhus-like illness.

With an infection that is so widespread it might be expected that infection would be transmitted in a simple way easily detected, but in man no natural means of infection is known other than congenital and this accounts for only a very few cases. For congenital infection to occur it is commonly held that the mother must become infected during pregnancy but it has been suggested that it can result from chronic infection in the mother.

Man's domestic animals have often been suspected and there is little doubt that persons in close contact with dogs or cats ill with toxoplasmosis can acquire infection from them through faeces, urine, or saliva. The observation that rodents become infected when they eat the flesh of animals dead of toxoplasmosis suggested that human infection might come from infected meat. Reference is then made to the contribution by Rawal dealt with in the accompanying abstract. If raw meat were responsible, infection

might be expected, by anology with trichinellosis, to be commoner in women than in men. In fact the prevalence is the same in each sex.

The way, or more probably the ways, in which toxoplasmosis is spread have yet to be found. Toxoplasma have been found in the saliva of a child. It should not be forgotten that the animal with which man is most in contact is man.

G. F. B.

White Muscle Disease (Myopathy) in Lambs and Calves. VI.—Effects of Selenium and Vitamin E on Lambs. MUTH, O. H., OLDFIELD, J. E., SCHUBERT, J. R., & REMMERT, L. F. (1959). *Amer. J. Vet. Res.* **20.** 231.

In a small experiment involving groups of 12 sheep, addition of 0.1 p.p.m. selenite to the diet prevented the development of white muscle disease in lambs from ewes which were on a diet which normally produced the condition. Vitamin E given orally or parenterally to the ewes failed to protect the lambs. G. B. S. H.

Microbial Agents Associated with Pneumonia in Lambs. HAMBY, *et al.* (1959). *Amer. J. Vet. Res.* **20.** 87.

Past. haemolytica was present in the throats (by swab culture) of 25 of 41 lambs and 18 of 36 dams. *P. multocida* was recovered from a further 3 lambs and 5 dams. Two of the dams had mastitis associated with *P. multocida*, and the authors suggest that infection may have been from the lambs throats.

Of these 41 lambs, 14 showed lesions of pneumonia at slaughter and 10 of these 14 had given pasteurella throat swabs during early life.

Bacteriological examination of the lungs of 51 lambs which showed lesions of pneumonia yielded *P. multocida* from 15, *P. haemolytica* from 9, streptococci and micrococci from 7, staphylococci from 3, corynebacterium from 2, Pseudomonas from 5, and PPLO from 2.

A virus was recovered from pneumonic lesions in 1 of 8 other lambs. G. B. S. H.

Virus Associated with Lamb Pneumonia. HAMDY-SANGER. (1959). *Amer. J. Vet. Res.* **20.** 84.

Using pneumonic lesions from a slaughtered lamb, a virus was propogated in sheep kidney tissue culture. Inclusion bodies developed in the cytoplasm by the seventh day but the cells were not damaged.

Tissue cultures regularly produced pneumonia in mice and chick embryo died after inoculation.

G. B. S. H.

***Septicemic Pasteurellosis* in Lambs.** BIBERSTEIN, E. L., & KENNEDY, P. C. (1959). *Amer. J. Vet. Res.* **20.** 94.

A disease of sheep which conforms to the description of "haemorrhagic septicaemia" was studied. *Past. haemolytica* was regularly recovered from a variety of lesions and the stains were pathogenic for mice and guinea-pigs. Attempts to reproduce the disease in lambs failed except in cases where the inoculum was big and intravenous G. B. S. H.

The Effect of Intravenous Glucose Injections on Appetite in Adult Ewes. MANNING, R., ALEXANDER, G. I., KRUEGER, H. M., & BOGART, R. (1959). *Amer. J. Vet. Res.* **20.** 242.

Intravenous infusion of from 1.67 to 8.3 grammes glucose per kilogram of bodyweight during 2-hour periods did not affect the food consumption or feeding behaviour of ewes. G. B. S. H.

A Succinoxidase Inhibitor in Feeds Associated with Muscular Dystrophy in Lambs and Calves. CARTAN, G. H., & SWINGLE, K. F. (1959). *Amer. J. Vet. Res.* **20.** 235.

A factor which inhibits the succinoxidase system was extracted from hay. It was absent from growing hay but appeared during the curing process and prolonged curing appeared to increase the amount. The inhibitory activity was partly reversed by addition of *α-tocapherol* to the system and evidence is presented which suggests that muscular dystrophy in lambs and calves may be due to excessive amounts of succinoxidase inhibitor in the diets of their dams. G. B. S. H.

Sensitivity Testing of 200 Strains of Salmonella to Furazolidone. MANN, P. H. (1958). *Poult. Sci.* **37.** 1,468-9.

Two hundred freshly isolated strains of Salmonella, belonging to 19 different serotypes, were tested *in vitro* for sensitivity to furazolidone. All were sensitive.

E. A. G.

Closed-circuit Halothane Anaesthesia in the Dog. LUMB, W. V. (1959). *J.A.V.M.A.* **134.** 218-21.

A brief review of the literature on halothane is followed by a report of the use of this anaesthetic agent in 117 dogs, 14 of which were anaesthetised twice; 4, three times and 1, four times.

Various combinations of pre-anaesthetic and anaesthetic drugs were employed and with one exception anaesthesia was always induced with a barbiturate. Endotracheal intubation was then performed and the halothane was administered from a Foregger anaesthetic apparatus in a closed circuit with carbon dioxide absorption. In most cases anaesthesia was maintained for more than one hour, and an anaesthetist was in constant attendance throughout.

Apnoea frequently developed, but was easily reversible. The dogs had hyperaemic mucous membranes but there was no excessive haemorrhage in the operative field. Four dogs showed signs of surgical shock. Muscle relaxation was adequate for major abdominal and thoracic surgery. Recovery from anaesthesia was usually rapid and quiet.

One death occurred early in the series of cases before the potency of the drug was fully appreciated.

(*Abstractor's note:* Numbers in the text indicate that this article should be followed by a list of 14 references, but this list does not appear.)

L. W. H.

News and Comment

THE NUTRITION SOCIETY

At a recent meeting of the Nutrition Society Mr. A. L. Bacharach was elected President in succession to Dame Harriette Chick, D.B.E. Professor R. G. Baskett, O.B.E., Mr. F. le Gros Clark, Dr. H. N. Munro, and Dr. F. Yates, F.R.S., were elected Ordinary Members of Council. Dr. R. J. L. Allen of Beecham Foods Ltd., Brentford, Middlesex, is Hon. Secretary.

MINISTRY OF AGRICULTURE VETERINARY FIELD STAFF

The Ministry of Agriculture Animal Health Division reports that Mr. G. A. Plummer, Veterinary Officer, who was stationed at Newcastle upon Tyne, has resigned.

THE SUPPLEMENT

Will honorary secretaries of Divisions kindly note that the current number of THE SUPPLEMENT has closed for press. Assuming that the present difficulties in the printing trade do not increase, it is hoped to publish THE SUPPLEMENT on June 27th.

THE ROYAL COLLEGE OF VETERINARY SURGEONS

In a ceremony at the XVIth International Veterinary Congress in Madrid, on Thursday, May 21st, the President, Professor T. J. Bosworth, presented the Diploma of Honorary Associate of the College—which is the highest honour the College can award—to the following persons:—

Professor E. C. Amoroso (Great Britain),
Professor P. Carda (Spain),
Professor C. Sanz Egana (deceased) (Spain),
Dr. K. V. L. Kesteven (FAO, Rome),
Professor L. Seekles (The Netherlands).

R.C.V.S. DAY

An exceedingly successful R.C.V.S. Day was held last Tuesday, June 2nd. The Service of Commemoration of Benefactors was held in the Queen's Chapel of the Savoy, being conducted by the College Chaplain, the Rev. C. L. Cresswell, K.C.V.O. The sermon was preached by the Rev. G. A. Willis, B.V.SC., M.R.C.V.S.

The Service was followed by the Annual Luncheon, at which the principal guest was H.R.H. The Duke of Gloucester, K.G., Honorary Associate of the College, who proposed the toast of the College. The President, Professor T. J. Bosworth, replied. The toast of the guests proposed by Dr. Guy Anderson was replied to by the President-Elect of the American Veterinary Medical Association, Dr. S. F. Scheidy.

Amongst those present were the Irish Ambassador, Lord Merthyr, Lord Lucas of Chilworth, Lord John Hope, M.P., Mr. H. F. Hebeler, President B.V.A., Mr. D. F. Oliver, Hon. Secretary of the B.V.A. Mr. P. H. Collick, M.P., Sir Anthony Hurd, M.P., Sir Wavell Wakefield, M.P., Sir James Duncan, M.P., Mr. A. J. Champion, M.P., Commander J. F. W. Maitland, M.P., Sir James Henderson-Stewart, M.P., Sir Jocelyn Lucas, M.P., Mr. A. Albu, M.P., Wing Com. R. Grant-Ferris, M.P., Mr. F. T. Willey, M.P., Mr. George Brown, M.P., Mr. T. F. Peart, M.P., Sir Charles Cunningham, Sir Hilton Poynton, Sir Paul Sinker, Sir Keith Murray, Sir Douglas Logan, Sir Geoffrey Marshall, Sir Cecil Wakeley, Sir Miles Clifford, Sir Henry Dale, Sir Charles Lovatt Evans, Sir Alick Buchanan-Smith, Dr. A. J. McConnell and Mr. W. G. Agnew.

At the Annual General Meeting held in the afternoon at the Royal College, the following gentlemen were declared elected to the Council, following the recent ballot: Professor Lamont, Dr. W. R. Wool-

Our photograph shows Professor E. C. Amoroso receiving the Diploma of Honorary Associate from the President of the Royal College, Professor T. J. Bosworth.

dridge, Mr. J. B. White, Mr. Edward Wilkinson, Mr. Harry Holroyd and (for one year) Mr. H. M. Wilson.

A full account of R.C.V.S. Day will be published next week, if the present difficulties in the printing industry permit.

GLASGOW UNIVERSITY VETERINARY MEDICAL ASSOCIATION

The Earl of Naresborough was the Presiding Patron at G.U.V.M.A.'s eighth annual rodeo and gymkhana held on May 23rd. The ring entry was good and the judge commended the standard of the jumping.

Eight donkeys came under starter's orders for the staff " donkey derby " which was telerecorded. Other attractions included a pipe band, children's pet show and side shows. It is estimated that a cheque for £150 will be presented to the Guide Dogs for the Blind Association. The Committee thank firms associated with the profession who responded to the appeal by sending donations and prizes.

THE JOINT MEASUREMENT SCHEME

At the Newark Show on May 9th, the 12. 2h.h. show pony " Arden Bronze " was objected to on grounds of height and when measured by two referees appointed by the Royal College of Veterinary Surgeons was found to be over height for its class and the objection was, therefore, upheld. " Arden Bronze " is a five-year-old chestnut pony owned by Mrs. G. Spooner. It is now ineligible to compete in classes " not exceeding 12. 2h.h."

THE REGISTER OF VETERINARY SURGEONS

The names of the following members have been removed from the Registers under Section 25 (3), Veterinary Surgeons Act, 1948:—

1. *Register of Veterinary Surgeons*

AUDEBERT, Henri Gabriel, France, May 7th, 1959.
LINTON, George Alexander, U.S.A., May 7th, 1959.
MCNELIS, Neil Joseph, Australia, May 7th, 1959.
*RICHARDSON, Ulick Forster, S. Africa, March 18th, 1959.
TUFTS, Fergus Andrew, U.S.A., May 7th, 1959.
WHITE, John Herman Munro, Australia, May 7th, 1959.

2. *Commonwealth and Colonial List*

*WILSON, Margaret Eleanor (Mrs. Evans), Australia, March 18th, 1959.

3. *Supplementary Veterinary Register*

*PICKWORTH, William Henry, Walsall, March 18th, 1959.

Registration in the Commonwealth and Colonial List

CLATWORTHY, Richard Hugh, B.V.SC. (PRETORIA), c/o Rhodesia House, 429, Strand, London, W.C.2, May 28th, 1959.

* Indicates that name removal is at own request.

UNIVERSITY NEWS

Liverpool

Professor J. G. Wright, Dean of the Faculty of Veterinary Science, and Professor of Veterinary Surgery, will be visiting the Faculty of Veterinary Medicine, University of Giessen, Germany, during July, 1959, under the Foreign University Interchange Scheme.

At its Meeting held on May 26th, 1959, the Council of the University approved the appointment, as from October 1st, 1959, of Dr. F. T. W. Jordan, Lecturer in Veterinary Preventive Medicine, as Senior Lecturer in Veterinary Preventive Medicine.

Belfast

At a meeting of the Faculty of Agriculture held on Monday, May 25th, Professor H. G. Lamont was unanimously elected Dean of the Faculty for the academic year 1959-60.

THE COLONIAL VETERINARY SERVICE

New appointments. A. C. Bygrave, B.V.SC., Veterinary Officer, Tanganyika; P. K. Van Stellingwerff, B.V.SC., Veterinary Officer, Western Region of Nigeria.

Promotion. W. W. Kirkby, M.B.E., Senior Veterinary Officer, Northern Nigeria, becomes Principal Veterinary Officer (Tsetse Control), Northern Nigeria.

PERSONAL

Colonel George Barnett, O.B.E., M.C., has been appointed Colonel Commandant, Royal Army Veterinary Corps, in succession to Brigadier J. J. Plunkett, C.B.E.

Births

HATCH.—On May 9th, 1959, at the Warren Maternity Home, Abingdon, Berks., to Jean (*née* Harrison), wife of John Hatch, B.A., B.SC. (VET.), M.R.C.V.S., a son, Alan Stuart.

LOFTUS.—On April 28th, 1959, to Evelyn, wife of Anthony Cole Loftus, M.R.C.V.S., of Coogee, N.S.W., Australia, a son, John Ernest, brother for Richard and Susan.

MCCORMICK.—On May 26th, at Crosshaven, Erleigh Road, Reading, to Rita (*née* Nunn), wife of Jim McCormick, M.R.C.V.S., a second daughter, Angela Rachel Carlisle.

PHILLIPS.—On April 23rd, 1959, at The City Hospital, Exeter, to Diana, wife of D. H. Phillips, B.V.SC., M.R.C.V.S., a daughter, Mary.

SUTHERLAND.—On May 22nd, 1959, to Jill, wife of D. J. Bruce Sutherland, M.R.C.V.S., 81, Benton Street, Hadleigh, Suffolk, a daughter, Shelagh, sister for Helen.

TUCKETT.—On May 26th, at The Gables Nursing Home, Aylesbury, to Betty, wife of Paul Tuckett, M.R.C.V.S., a son, Alan Edwyn.

COMING EVENTS

June

10th (Wed.). Meeting of the Society for the Study of Animal Breeding in the West Hall of the Royal Society of Medicine, 1, Wimpole Street, London, W.1, 11.30 a.m.

11th (Thurs.). Annual Meeting of the R.A.V.C. Golfing Society at the North Hants Golf Club, Fleet, 10 a.m.

12th (Fri.). Annual Dinner of the R.A.V.C. Officers' Club at Grosvenor House.

Annual General Meeting of the Shropshire Veterinary Club at the Woodlands Country Club, Wroxeter, Salop, 8 p.m.

13th and 14th (Sat. and Sun.). 7th Annual Reunion and Dinner of the R.V.C. '52 Club at Streatley.

17th (Wed.). Annual General Meeting of the North of Scotland Division in the Northern Hotel, Aberdeen, 2.30 p.m.

3rd Annual General Meeting of the B.S.A.V.A. Metropolitan Region at the Royal Society of Medicine, 6 p.m.

18th (Thurs.). Meeting of the Lakeland Veterinary Association at the Shap Wells Hotel, Shap, 6 p.m.

27th (Sat.). Meeting of the B.S.A.V.A. North West Region at the Brocket Arms, Wigan.

30th (Tues). Summer Meeting of the A.V.T. & R.W.s (Northern Region) at Aberystwyth, 11.30 a.m.

July

1st (Wed.). Meeting of the B.V.A. Overseas Committee at 7, Mansfield Street, London, N.W.1, 2 p.m.

8th (Wed.). Summer Meeting of the Lancashire Veterinary Association at Nantwich, 2.30 p.m. Reception and dinner at the Swan Hotel, Tarporley, 6.30 p.m. for 7 p.m.

August

30th to 5th September. 77th Annual Congress of the British Veterinary Association in Folkestone. (Official opening Monday, August 31st.)

B.V.A. COMMITTEE AND COUNCIL MEETINGS IN LONDON

Tuesday, July 14th—at 7, Mansfield Street, W.1.
4.30 p.m. Finance Sub-committee.
Wednesday, July 15th—at 7, Mansfield Street, W.1.
10.00 a.m. Veterinary State Medicine Committee.
11.30 a.m. Home Appointments Committee.
2.15 p.m. Small Animals Committee.
Thursday, July 16th—at 7, Mansfield Street, W.1.
10.00 a.m. Parliamentary and Public Relations Committee.
12 noon Farm Livestock Committee.
2.15 p.m. General Purposes and Finance Committee.
Friday, July 17th—at Connaught Rooms, Great Queen Street, W.C.2.
10.15 a.m. Council Meeting.

FOOT-AND-MOUTH DISEASE

Restrictions on the movement and marketing of livestock on the Isle of Wight, resulting from outbreaks of foot-and-mouth disease at Cheverton Slaughterhouse and Wootton Farm, Wootton, Newport, Isle of Wight, were withdrawn as from midnight on May 28th.

A further outbreak of the disease has, however, been confirmed in Wootton among cattle, and an Area extending for about 5 miles around the infected premises has been declared to be an Infected Area.

Animals may not be moved in the Area without a licence and the holding of markets in the Area is restricted.

R.C.V.S. PRIZES

At the Annual General Meeting of the Royal College the following honours and prizes were awarded :—

Fellowship by Election, Mr. G. N. Gould, J.P. ; Honorary Associate, Dr. J. Hammond, C.B.E., F.R.S. ; John Henry Steel Memorial Medal, Dr. R. E. Glover, M.A. ; Francis Hogg Prize, Miss J. O. Joshua ; R. W. Hall, of Barry, Glamorgan, Prize, Mr. J. W. Bruford; FitzWygram Prize, Mr. D. B. Ross, B.A. ; Williams, Walley and Lawson Prize, Mr. M. A. E. Rex, B.A. ; Fellowship by Examination, Professor J. F. A. Sprent, PH.D., B.SC.

DR. HAMMOND ON TWINNING

A solution to Britain's problem of producing more beef would be to produce twin calves, Dr. J. Hammond suggested when he addressed the seventh annual general meeting of members of Buxton Cattle Breeding Centre on May 26th. He believed that by inducing twins artificially, and feeding for that purpose, the difficulties associated with twinning in cattle could be overcome. If the experiments now in progress were successful the method might eventually be applied to all A.I. stations.

Speaking of the possible introduction of Charollais cattle to this country, Dr. Hammond said he would like to see them brought in and put on Ayrshires. They would give good weight for age, and that was another way in which to increase the beef supply. If the British farmer wished to go out and capture the beef market, he had a wonderful opportunity at the present time. Dr. Hammond expressed the view that it would be three years before the world supply of beef would return to normal—it might be longer.

" THE VETERINARY RECORD "

Our readers will be aware of a serious dispute in the printing industry which, after this week, may become more acute. We ask their indulgence in case of late delivery or any diminution in size of subsequent issues, which will be due to factors quite outside our control.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Dumfries. Tinwald Parks, Amisfield (May 28).
Glouces. Maes Knoll Farm, Norton Malreward, Pensford (June 1).
Lancs. New Manor Farm, Preston Brook, Warrington (June 1).
Stirling. Easter Blairskaith Farm, Baldernock (June 1).

Sussex. Catts Green Farm, Staplecross, Robertsbridge (May 29).

Foot-and-Mouth Disease

Isle of Wight. Woodside Farm, Wootton, Newport (May 29).

Fowl Pest

Essex. Finchleys, Dansey Lane, Little Waltham, Chelmsford; Green End, Great Leighs (May 27); Cheese House Farm, Great Leighs; Boscombe, Boreham Road, Little Waltham, Chelmsford (May 28); Applegold Cottage, Church Lane, Little Leighs, Chelmsford; Mount Mascalls Farm, Boreham; Crees Farm Nurseries, School Lane, Great Leighs, Chelmsford; White City Garden, Great Leighs (May 29); Blasford Hill, Little Waltham; Hawkins Farm, Cock Clarks, Purleigh; Leylands Farm, Terling (May 30); Ayletts Farm, Broomfield; 2, Church Road, Boreham; 36, Castle Close, Great Leighs; Gubbins Hall, Great Leighs, Chelmsford (June 1).

Herts. Lynch Lane, Fowlmore, Royston (May 28).

Lancs. Isherwood Farm, Walshaw, Bury (May 26); Temple Farm, Edgworth, Bolton; Highbarn Farm, Harwood (June 1).

Surrey. Ilfrators, Selsdon Vale, Selsdon (May 28); Quantocks, Selsdon Vale, Selsdon; Oakfield, Woodland Gardens, Selsdon (June 1).

Yorks Peterwood Farm, Fulwood, Sheffield, 10 (May 29).

Swine Fever

Ches. Oxheys, Rushton, Tarporley (May 28); Ivy House, Moreton, Wirral (June 1).

Devon. Clapperdown Farm, Bondleigh, North Tawton (May 28); Pontins Barton Hall, Chalet Hotel, Torquay; Ryden House, Kingsteignton, Newton Abbot (June 1).

Dorset. R.A.C. Centre, Bovington Camp, Wareham (May 26).

Essex. The Mill, Great Oakley, Harwich (May 29).

Glam. Lower House Farm, Colwinston, Cowbridge (June 1).

Gloucs. Blacksmith's Meadow, Fiddlers Green, Cheltenham (May 26); Myrtle Cottage, New Passage, Pilning, Bristol; Bellamys Farm, Longney (May 29); Maulls Nursery, Hambrook, Bristol (June 1).

Lancs. Higher Rhoden Farm, Oswaldtwistle, Accrington; Union Bank Farm, Northwich Road, Dutton, Warrington (May 26); Old Ship Farm, Delf Lane, Kirkby, Liverpool (May 29).

Norfolk. Freehold Allotments, Bure Road, Runham Vauxhall, Great Yarmouth; Street Farm, Denton, Harleston (June 1).

Monmouth. Home Farm, St. Cadoc's Hospital, Caerleon, Newport (May 28).

Northants. 41, Harringworth, Corby (May 28); Manor Farm, Syresham, Brackley; 252, Newton Road, Rushden (June 29).

Staffs. North Hartley Green Farm, Gayton (June 1).

Suffolk. Limes Farm, Rushmore St. Andrew, Ipswich (May 27).

Surrey. Woodside, Selsdon Vale, Sanderstead (May 26).

Wilts. Park Farm, Corsham (June 1).

Worcs. Hatfield Villa, Norton (May 28).

Yorks. 102, Keresforth Hill Road, Kingstone, Barnsley (June 1).

AN EXHIBITION AT THE R.C.V.S.

An interesting exhibition illustrating the beginnings of veterinary education in England was staged in the library of the Royal College during the past week. Members who were in London for R.C.V.S. Day were able to see a unique display of valuable books and old prints, together with some original manuscripts which were on loan from the library of the Royal Veterinary College and the Wellcome Historical Library.

Professor L. P. Pugh was responsible for the lay-out of the show cases whose contents depicted the early history of veterinary science in this country, the foundation of the Odiham Agricultural Society, and the personalities behind the establishment of the Veterinary College in London. Among the many interesting exhibits in this section, the original minute book of the Odiham Agricultural Society, and possibly the only remaining print of the original St. Bel "Plan" of 1790 were notable. Both these items had come back into veterinary hands after many years in oblivion. The Odiham Minute Book had been traced by Mr. W. G. R. Oates, Registrar of the R.C.V.S. and Miss Raymer, the Librarian, and Professor Pugh had been responsible for locating the St. Bel "Plan" in the West Country.

Another section of the exhibition, referred to in our columns last week, was planned by Mr. J. W. Barber-Lomax and illustrated Moorcroft's journey into Hiundes. Three prints were on view from original water-colour drawings in the India Office and depicted scenes from Moorcroft's travels. There were also early prints and maps on view, and all the exhibits were clearly and spaciously set out with explanatory notes.

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

The R.C.V.S. Council Elections

Sir,—May I by your courtesy express my thanks to those who have elected me to the Council of the Royal College of Veterinary Surgeons and say that it will be my endeavour to justify the confidence they have placed in me.

Yours faithfully,
J. B. WHITE.

The Spinney,
63, Bouverie Avenue,
Salisbury.
June 2nd, 1959.

A Matter of Opinion

Sir,—The words of the secretary of the A.R.C. carry the weight and authority of the holder of high office, so it is with some hesitation that I suggest there might be grounds for challenging one of Sir William Slater's statements made in his January lecture at Bangor (*Vet. Rec.* **71.** 371). In the introduction he is reported as saying: "If Share-Jones could walk around the laboratories and farms of this College he would be greatly heartened to see the outstanding work on animal husbandry, physiology, and

biochemistry in progress, with which my Council is glad to be associated." Only in the belief that the development at Bangor was a nucleus for his desired Welsh veterinary school could I believe that Professor Share-Jones would have been heartened; as a veterinarian he would have argued with all the skill and fervour he possessed in such marked degree that a veterinary school was the obvious and logical location for such an enterprise as it offered the most favourable environment for the proper study of farm animal problems, both fundamental and applied.

This is a matter of extreme importance to the profession and ultimately to the country, as it would appear unlikely that our Schools can attain their full stature so long as official policy is to establish and maintain dispersed units of which Bangor is but one example.

Yours faithfully,
JOHN C. GRANT.

Veterinary Department,
Marischal College,
Aberdeen.
May 27th, 1959.

Suspected Bovine Malignant Catarrh

Sir,—Referring to the clinical report (page 191 of your issue of March 7th), may I, since no one else has taken up the point, suggest that a diagnosis of malignant catarrh in bovine animals need not remain a matter of suspicion or conjecture, when histological examination of sections of brain, liver, kidney and lung will reveal the pathognomonic massive round-celled perivascular and periportal infiltrations in positive cases.

Moreover, the causal agent is readily transmitted to rabbits. Cases are not uncommon in Syria and Lebanon, and on each of four occasions when rabbits have been inoculated transmission has been effected.

Yours faithfully,
R. DAUBNEY.

Food and Agriculture Organisation of the United Nations,
Mission to Lebanon,
Beirut,
B.P. 3216.
May 21st, 1959.

Bracken Poisoning in Pigs

Sir,—We suspect that we have had some recent cases of poisoning by bracken in pigs, and would be very grateful if any of your readers with similar experiences would communicate with us.

Yours faithfully,
H. C. SWANN,
J. C. BARROWMAN.

Veterinary Centre and Hospital,
London Road,
King's Lynn,
Norfolk.
May 27th, 1959.

Intussusception in a *Chinchilla lanigera*

Sir,—It was refreshing to see the case report by Brown and Morgan (*Vet. Rec.* **71.** 430) describing intussusception in a female *Chinchilla lanigera*. It is important that veterinary surgeons should note the versatility of their colleagues.

Intravenous anaesthesia such as they describe is successful for a wide variety of smaller creatures but, as they have indicated, it is essential that such intraperitoneal anaesthetics as may be used should be diluted at least with an equal volume of sterile water. The authors may be interested to note that subcutaneous injection of similar agents can produce quite successful anaesthesia and is sometimes easier.

The whole problem of chinchilla maintenance in this country is one which requires urgent attention by the veterinary profession. Such urgency is not admitted when one realises that the publication of Brown and Morgan's contribution appears to be some months after it was written: in the paragraph headed "Sequelae" the authors say "subsequently she bred-back and is again due to litter during the first week in February." One presumes this meant February, 1959.

The authors state "it is suggested that the condition is more common in excitable individuals and that parasitism, obstructions, enteritis, and dietetic upsets are other predisposing factors." In this they are undoubtedly correct, but all the conditions they mention could be, and probably are, the result of "shock" syndrome as it applies to chinchillas. It should be remembered that whilst the chinchilla is a hardy animal, it is also very sensitive. One of the authors (D. H. W. B.) had recently imported direct from California 12 chinchillas; it was one of these, a female, which on arrival was said to exhibit abdominal discomfort. It is possible that this chinchilla, in common with many others, experienced during the previous 10 days the following major changes in its daily pattern:—

A journey from the chinchilla ranch in California to a rail head.
A journey on a train to an airport.
A journey from the airport across the Atlantic to Europe.
A journey from Europe to Great Britain.
Transport from the airport in Great Britain to a rail head.
A rail journey to a distributing agency.
A final journey from an agency to the owner.

The above list of journeys may not be true in all respects but it is a fair average which applies to those animals freshly imported. It must be remembered also that throughout these journeys the animal's surrounding temperatures vary considerably, there is considerable noise, diet may vary in quantity, quality, and timing, and the technique of handling and transporting cages may not always be the best. All this builds up the stress suffered by the chinchilla.

As I understand it there is a very heavy death rate in newly imported chinchilla and in chinchilla newly arrived at the purchaser's home. If clinical histories and *post-mortem* findings are to be accepted, it is clear that exhausted adrenals coupled with other evidence clearly underline the lethal effects of the "shock" syndrome in chinchilla—which effects may not be shown for some weeks. In the knowledge that a state of stress is easily induced in the chinchilla one questions the wisdom of the alternative methods of reduction suggested by Brown and Morgan for the reduction of intussusception. These

include vertical suspension of the anaesthetised animal by its tail, subsequent repeated introduction and withdrawal of a blunt glass rod, and the introduction of a plastic tube by which a saturated sugar solution can be introduced. They further note "one correspondent even suggests swinging the animal (presumably anaesthetised) in circles by its hind legs." Even if this vigorous system should rectify the intussusception I cannot believe it is in the best interest of the chinchilla to handle it in this fashion.

The authors are to be congratulated on the successful way in which they tackled a delicate surgical operation. We should all bear in mind, however, that the intussusception undoubtedly is the result of "stress" in the animal and should not be regarded as an isolated surgical disease.

Yours faithfully,
OLIVER GRAHAM-JONES,
Veterinary Officer.

The Zoological Society of London,
Regent's Park,
London, N.W.1.
May 28th, 1959.

Hypomagnesaemic Tetany

Sir,—Mr. H. C. Wilson in his letter to THE VETERINARY RECORD of May 16th has brought to notice 2 cases of interest upon which he invites comment. The following observations are offered:—

In the case of the Ayrshire cow showing signs of tetany the administration of magnesium was very reasonable in the light of the clinical evidence available, but we feel that the tetany must have been of non-hypomagnesaemic origin for the following reasons:—

(i) The cow was in its first lactation and as such it was not as susceptible to hypomagnesaemia as are older cows.

(ii) It was housed and fed a dairy ration. Hypomagnesaemia is not very often encountered in dairy cows on concentrate rations, especially when the concentrates are fortified with a high magnesium supplement as was the case in point. Hyper-excitability from other causes may, of course, be prevalent in some herds in winter.

(iii) Blood analysis showed a normal level of magnesium and of calcium.

It would have been interesting to have known the inorganic phosphorus, sugar, and ketone levels in the blood.

The recumbent ewe showing an increased magnesium level is very interesting in that it is unusual for sheep to consume voluntarily very large quantities of mineral mixtures. However, Stewart and Moodie (*J. comp. Path.* **66.** 10) have shown that serum magnesium levels in sheep can be raised very markedly by oral administration of magnesium salts. We presume, as Mr. Wilson does, that the excessive intake of magnesium and the consequent high serum magnesium level could lead to the recumbency.

The apparent anomaly in the treatment of calf No. 10 in our experiments reported on March 28th in THE RECORD is resolved if it is appreciated that there is not a consistent interaction between calcium and magnesium. For instance, it is known that a clinically normal animal may have a very low serum magnesium level with normal calcium and that this same animal may become clinically affected with tetany when the serum calcium falls. This is an instance of the narrowing of the calcium/magnesium ration being associated with signs of hyper-excitability whereas the instance of the ewe quoted by Mr. Wilson also showed a narrowing of the calcium/magnesium ratio but this time it was associated with signs of depression. In the former case the narrowing of the ratio was caused by a drop in the calcium level, and in the latter by an elevation of the magnesium level.

In the case of our No. 10 calf there were experimental reasons which made it undesirable to increase the total magnesium content of the calf's blood and tissues and in consequence magnesium was withheld. (In practice no such restrictions would apply.) In view of the possibility of a sudden drop in serum calcium precipitating clinical symptoms as mentioned in the previous paragraph the calcium was administered.

We would not agree with those veterinary surgeons who would treat hypomagnesaemia with calcium in preference to magnesium if, in fact, this means calcium alone rather than the Ca.Mg.P. complex. Whilst it is desirable to use calcium in the treatment of field cases of hypomagnesaemia we think that it is logical and reasonable to administer magnesium in addition.

Yours faithfully,
J. E. F. RANKIN,
J. R. TODD.

Ministry of Agriculture for Northern Ireland,
Veterinary Research Division,
The Farm,
Stormont,
Belfast.
May 29th, 1959.

Parasitic Otitis in Kittens

Sir,—An article which appeared in THE VETERINARY RECORD during 1958 (*Vet. Rec.* **70.** 740) relative to parasitic otitis in dogs stimulated me to examine a series of kittens presented for conditions other than otitis, chiefly castrations and spays.

Using an auroscope only, I examined 100 kittens which were presented consecutively and about which the owners made no complaint relative to "ear trouble," and found that 48½ per cent. had visible mites in their ears. One kitten had mites in one ear only (thus the ½ per cent.) and one had dead mites in one ear. This latter case had been treated by the owners as had one other negative case.

I do not know whether such a high rate of infestation in kittens is general but suspect that it probably is and is no doubt a great source of parasitic otitis in dogs.

Yours faithfully,
J. McCORMICK.

113, Oxford Road,
Reading.
June 1st, 1959.